Acute Pericarditis

The Pericardium

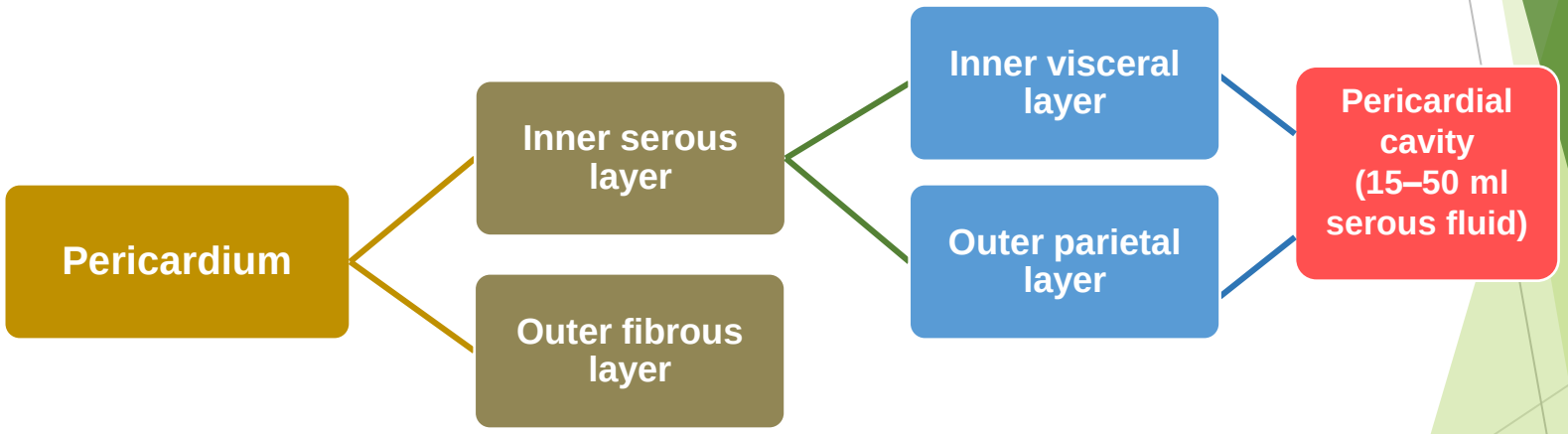
- A fibroelastic sac composed of visceral and parietal layers .
- It normally functions to protect the heart and reduce friction between the heart and surrounding organs.
- Both these layers are separated by a pericardial cavity.
- The cavity normally contains **15 to 50 ml** of straw-colored fluid.
- Visceral layer is in contact with the epicardium (ST elevation)

Pathophysiology of Pericardial Disease

IMS 350

Pericardium: Anatomy

- Thin, avascular, inelastic, flask-shaped sac
- Envelops the heart, proximal portions of the ascending aorta, pulmonary artery, pulmonary veins, and superior vena cava



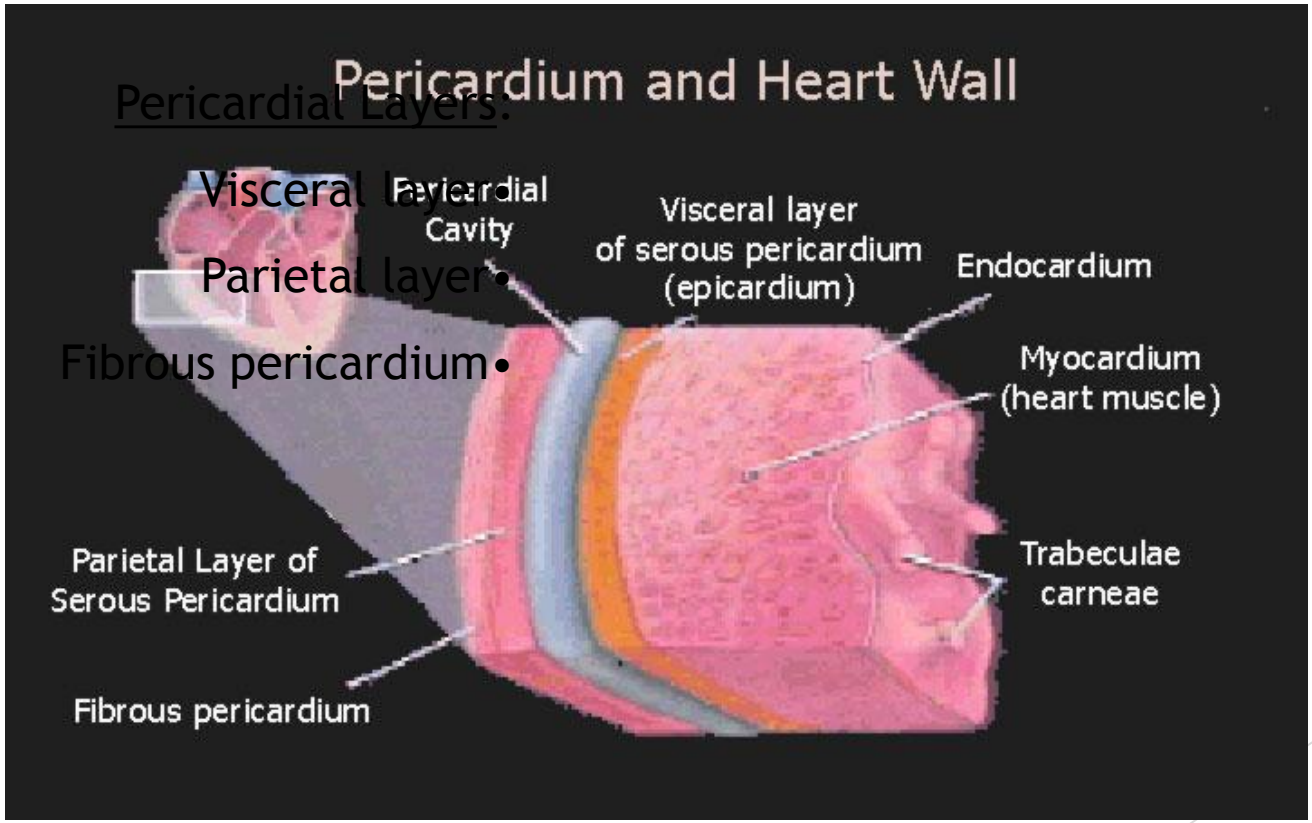
Pericardium - Anatomy

- Normal pericardium is a fibro-serous sac which surrounds the heart and adjoining portions of the great vessels.
- The inner visceral layer, also known as the epicardium, consists of a thin layer of mesothelial cells closely adherent to the surface of the heart. The epicardium is reflected onto the surface of the outer fibrous layer with which it forms the parietal pericardium.
- The parietal pericardium consists of collagenous fibrous tissue and elastic fibrils.
- Between the two layers lies the pericardial space, which contains approximately 10-50ml of fluid, which is an ultrafiltrate of plasma.
- Drainage of pericardial fluid is via right lymphatic duct and thoracic duct.

Pathophysiology of Pericardial Disease

IMS 350

Pericardium: Anatomy



Function of the Pericardium

1. Stabilization of the heart within the thoracic cavity by virtue of its ligamentous attachments -- limiting the heart's motion.

2. Protection of the heart from mechanical trauma and infection from adjoining structures.

3. The pericardial fluid functions as a lubricant and decreases friction of cardiac surface during systole and diastole.

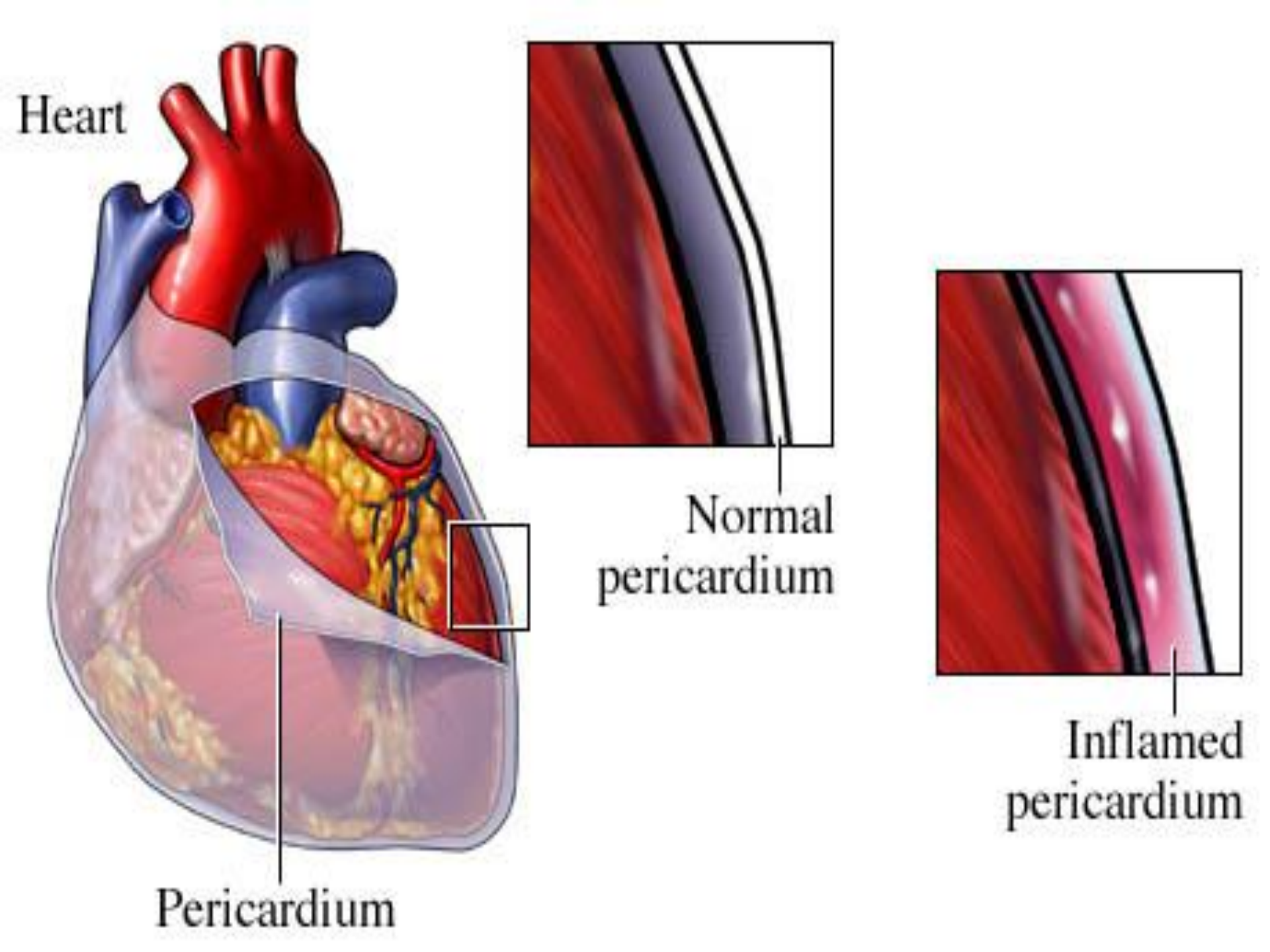
4. Prevention of excessive dilation of heart especially during sudden rise in intra-cardiac volume (e.g. acute aortic or mitral regurgitation).

Diseases of the Pericardium

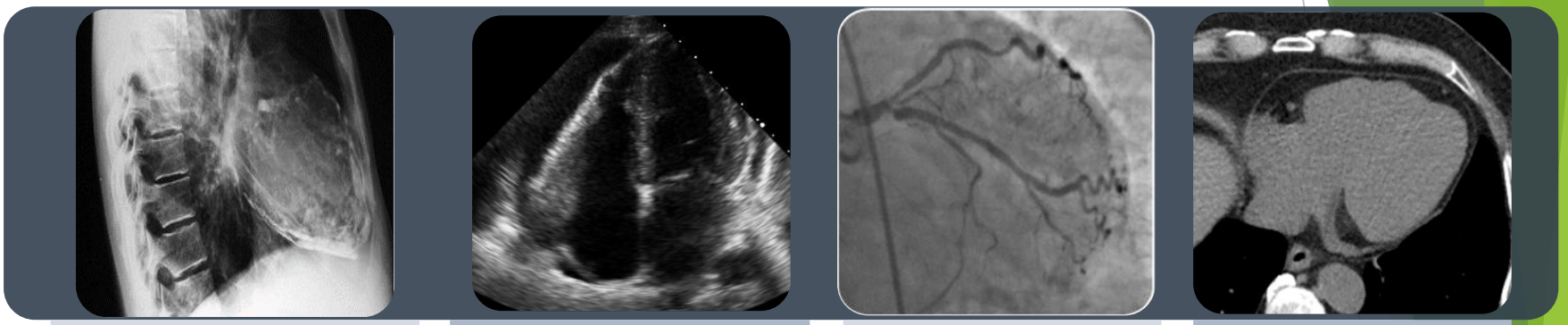
1) Acute and recurrent Pericarditis
2) Pericardial Effusion
3) Cardiac tamponade
4) Constrictive Pericarditis

Acute Pericarditis

- **inflammation of the pericardial sac**
- The term perimyocarditis is used for cases of acute pericarditis that also demonstrate myocardial inflammation.
- It may be accompanied by pericardial effusion, which is fluid accumulation in the pericardial sac.
- Can be an isolated entity or part of a systemic disease
- 0.1% of all hospitalized patients
- 5% of ER visits for chest pain without an MI


Heart
Pericardium
Normal pericardium
Inflamed pericardium

Pericardium: Imaging Modalities



Radiography

- Limited role
- Evaluation of calcification
- Enlarged cardiac silhouette

Echocardiography

- First-line imaging modality
- Effusion, thickening, constriction, and tamponade
- Function
- Limited field of view
- Operator- and imaging window–dependent

Cardiac Catheterization

- Measurements of ventricular pressures
- Constriction

Computed Tomography

- Pericardial calcification
- Thickening/effusion
- Retrospective electrocardiographically (ECG)–gated images can be obtained, although these are only for breath-held acquisitions

Causes of pericardial disease

Etiologies of Pericarditis

I. INFECTIVE

1. VIRAL - Coxsackie A and B, Influenza, adenovirus, HIV, etc.
2. BACTERIAL - Staphylococcus, pneumococcus, tuberculosis, etc.
3. FUNGAL - Candida
4. PARASITIC - Amoeba, candida, etc.

II. AUTOIMMUNE DISORDERS

1. Systemic lupus erythematosus (SLE)
2. Drug-Induced lupus (e.g. Hydralazine, Procainamide)
3. Rheumatoid Arthritis
4. Post Cardiac Injury Syndromes i.e. postmyocardial Infarction Syndrome, postcardiotomy syndrome, (Dressler's) etc.

III. NEOPLASM

1. Primary mesothelioma
2. Secondary, metastatic
3. Direct extension from adjoining tumor

IV. RADIATION PERICARDITIS

V. RENAL FAILURE (uremia)

VI. TRAUMATIC CARDIAC INJURY

1. Penetrating - stab wound, bullet wound
2. Blunt non-penetrating - automobile steering wheel accident

VII. IDIOPATHIC

Pathogenesis

1) Vasodilation:
→ transudation of fluid
2) Increased vascular permeability
→ leakage of protein
3) Leukocyte exudation
neutrophils and mononuclear cells

Pathology

depends on underlying cause and severity of inflammation
serous pericarditis
serofibrinous pericarditis
suppurative (purulent) pericarditis
hemorrhagic pericarditis

Clinical Features of Acute Pericarditis

Idiopathic/viral

* Pleuritic Chest pain
* Fever
* Pericardial Friction Rub

3 component:

a) atrial or pre-systolic component

b) ventricular systolic component (loudest)

c) ventricular diastolic component

* EKG: diffuse ST elevation
  PR segment depression

Idiopathic
In most case series, the majority of patients are not found to have an identifiable cause of pericardial disease. Frequently such cases are presumed to have a viral or autoimmune etiology.
Infections
Viral - Coxsackievirus, echovirus, adenovirus, EBV, CMV, influenza, varicella, rubella, HIV, hepatitis B, mumps, parvovirus B19, vaccina (smallpox vaccination)
Bacterial - Staphylococcus, Streptococcus, pneumococcus, Haemophilus, Neisseria (gonorrhoeae or meningitidis), Chlamydia (psittaci or trachomatis), Legionella, tuberculosis, Salmonella, Lyme disease
Mycoplasma
Fungal - Histoplasmosis, aspergillosis, blastomycosis, coccidiodomycosis, actinomycosis, nocardia, candida
Parasitic - Echinococcus, amebiasis, toxoplasmosis
Infective endocarditis with valve ring abscess
Radiation
Neoplasm
Metastatic - Lung or breast cancer, Hodgkin's disease, leukemia, melanoma
Primary - Rhabdomyosarcoma, teratoma, fibroma, lipoma, leiomyoma, angioma
Paraneoplastic
Cardiac
Early infarction pericarditis
Late postcardiac injury syndrome (Dressler's syndrome), also seen in other settings (eg, post-myocardial infarction and post-cardiac surgery)
Myocarditis
Dissecting aortic aneurysm

Trauma

Blunt

Penetrating

Iatrogenic - Catheter and pacemaker perforations, cardiopulmonary resuscitation, post-thoracic surgery

Autoimmune

Rheumatic diseases - Including lupus, rheumatoid arthritis, vasculitis, scleroderma, mixed connective disease

Other - Granulomatosis with polyangiitis (Wegener's), polyarteritis nodosa, sarcoidosis, inflammatory bowel disease (Crohn's, ulcerative colitis), Whipple's, giant cell arteritis, Behcet's disease, rheumatic fever

Drugs

Procainamide, isoniazid, or hydralazine as part of drug-induced lupus

Other - Cromolyn sodium, dantrolene, methysergide, anticoagulants, thrombolytics, phenytoin, penicillin, phenylbutazone, doxorubicin

Metabolic

Hypothyroidism - Primarily pericardial effusion

Uremia

Ovarian hyperstimulation syndrome

- Which of the following choices is the most common cause of acute pericarditis?

1) Neoplasm
2) Uremia
3) Viral/idiopathic etiology
4) Radiation exposure
5) Complication of myocardial infarction

- Which of the following choices is the most common cause of acute pericarditis?

1) Neoplasm
2) Uremia
3) Viral/idiopathic etiology
4) Radiation exposure
5) Complication of myocardial infarction

Diagnostic Clues

- History

  sudden onset of anterior chest pain that is pleuritic and substernal

- Physical exam

  Pericardial friction rub

- ECG

  most important laboratory clue

- ECHO

  Showed free area between pericardium and wall of L. ventricle(not useful in early stage of dry pericarditis)

Presence of at least two of the above features is necessary to make the diagnosis

Clinical Finding

❖ **Depond on the:**

- Type of inflammation.
- Sevirety of inflammation.
- Formation of pericardial fluid.

1. Dry pericarditis.
2. Percardial effusion without cardiac temponade.
3. Cardiac temponade.
4. Pericardial constriction.

1- Dry pericarditis

1- Chest Pain

❖Common characteristics 95%

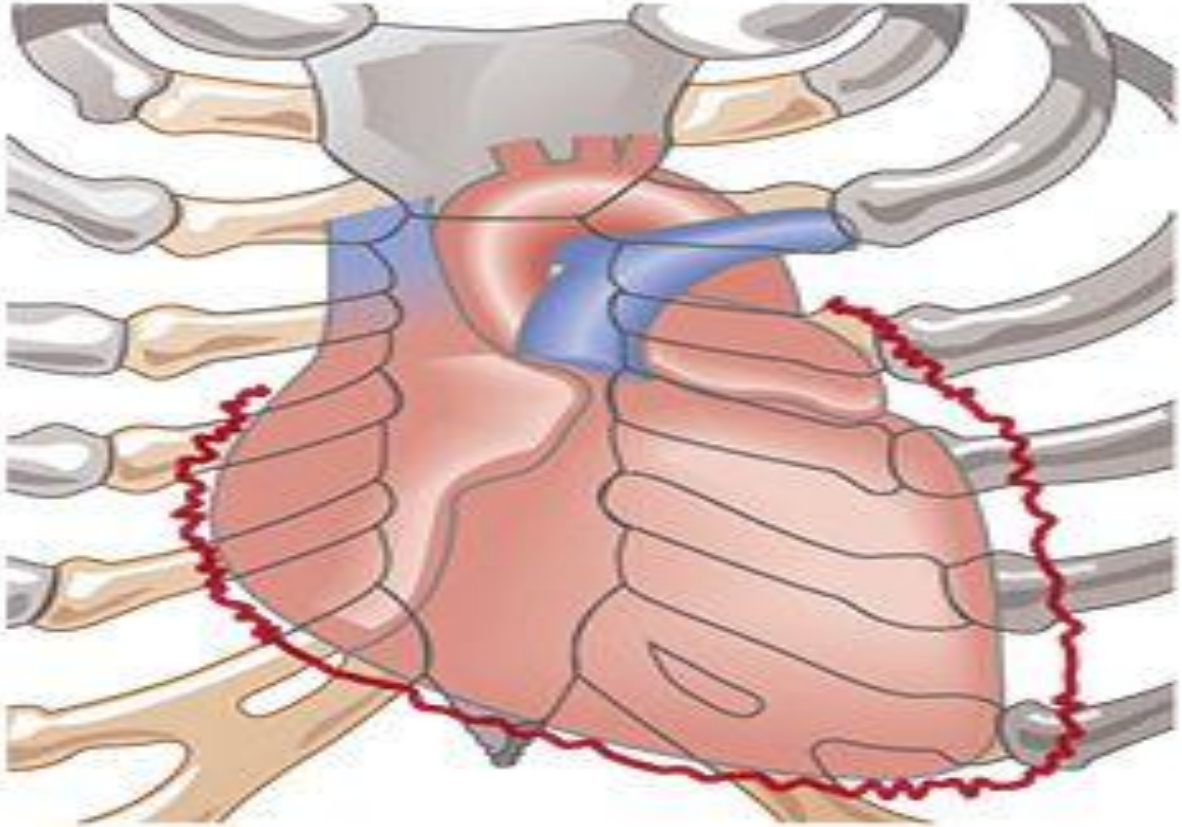
- retrosternl or precordial with raditaion to the neck, back, shoulder or arm
- sudden in onset

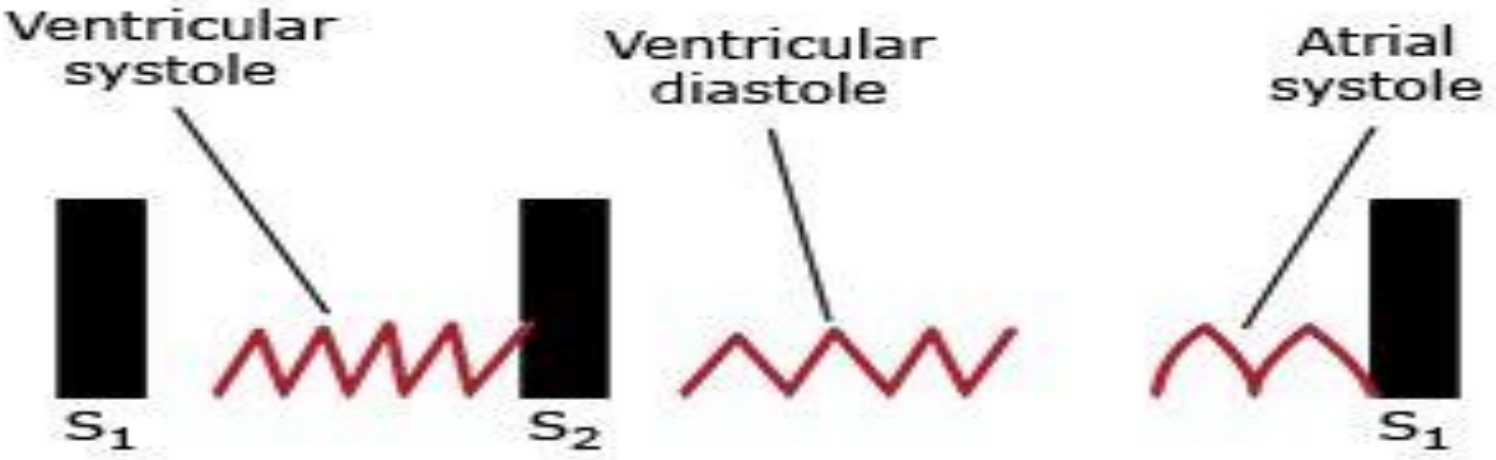
❖ Special characteristics

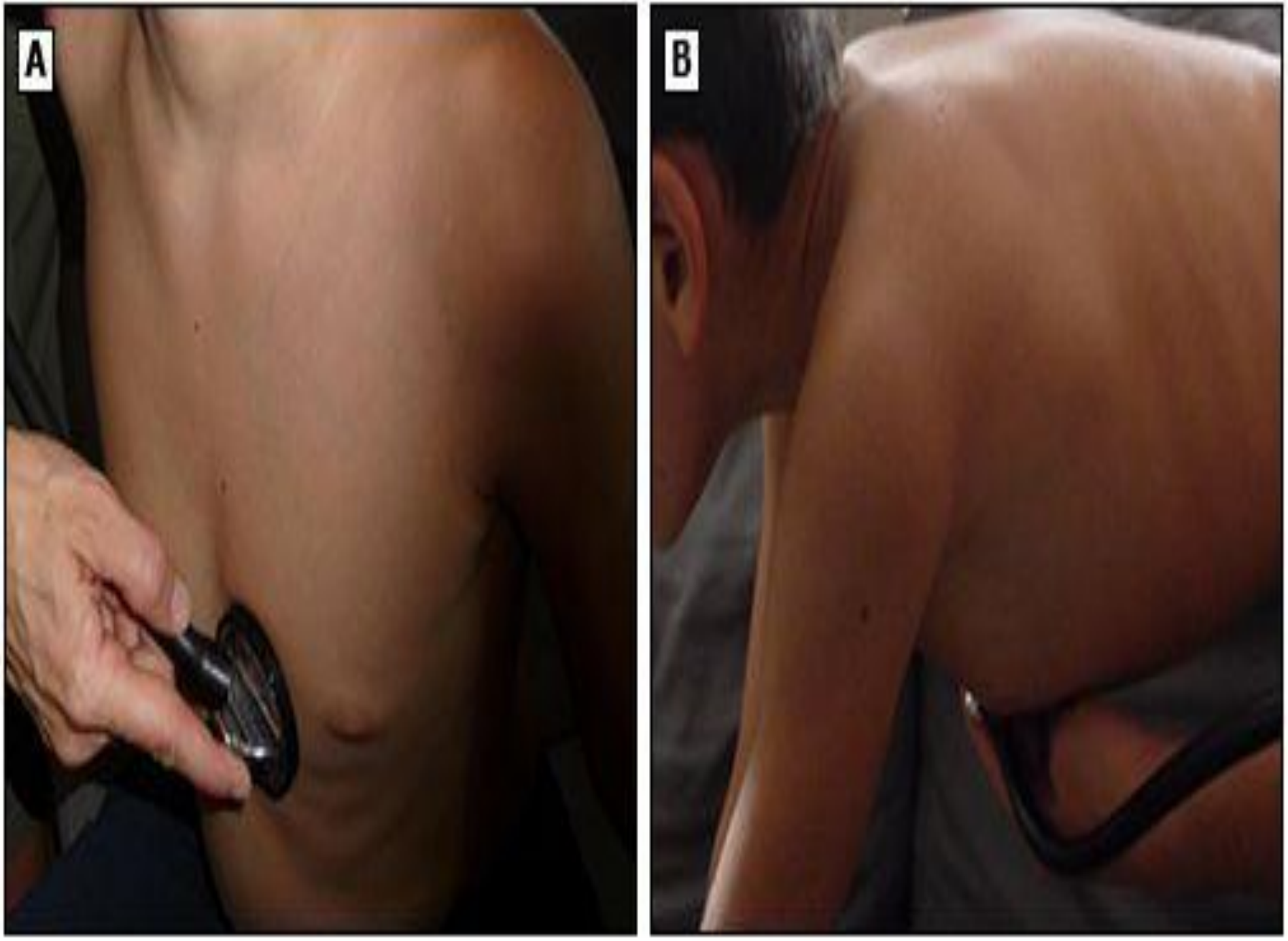
1) more likely to be sharp and pleuritic
2) Radiation to one or both trapezius muscle ridges, suggests a probable pericarditis .
3) worse with coughing, inspiration, swallowing
4) worse by lying supine, relieved by sitting and leaning forward

2- Pericardial friction rub

- is pathognomic for pericarditis
- Present in 85% of cases of pericarditis
- scratching or grating sound
- best heard with the diaphragm at the LSB with the patient leaning forward.
- Pericardial friction rub is audible throughout the respiratory cycle, whereas the pleural rub disappears
- Has 3 components, which correspond to ***atrial systole, ventricular systole, and early diastole.***
- Pericardial rub start to disappear when effusion develops




Ventricular systole
Ventricular diastole
Atrial systole
S1
S2
S1


A
B

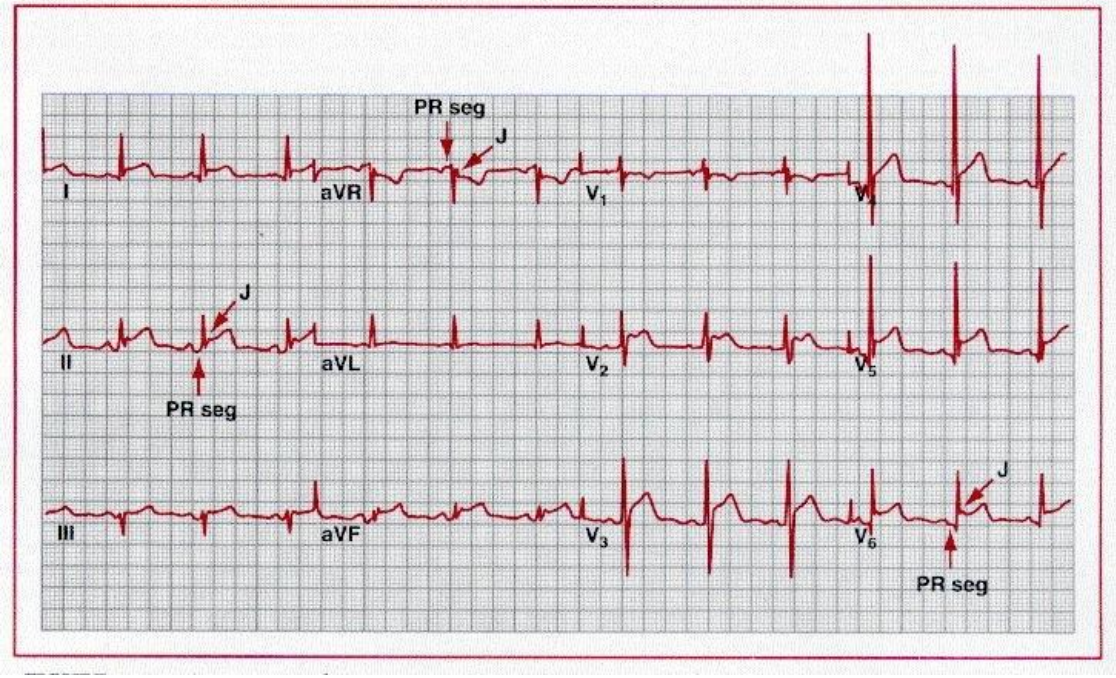
3- EKG

- Changes in (ECG) in patients with acute pericarditis signify inflammation of the epicardium .
- Widespread upward concave ST-segment elevation and PR-segment depression
- If the ratio of ST-segment elevation to T-wave amplitude in V6 > 0.24, acute pericarditis is almost always present.
- The EKG changes have 4 phases during the course of illness
- sustained arrhythmias are uncommon in acute pericarditis
- the presence of atrial or ventricular arrhythmias is suggestive of concomitant myocarditis or an unrelated cardiac disease.

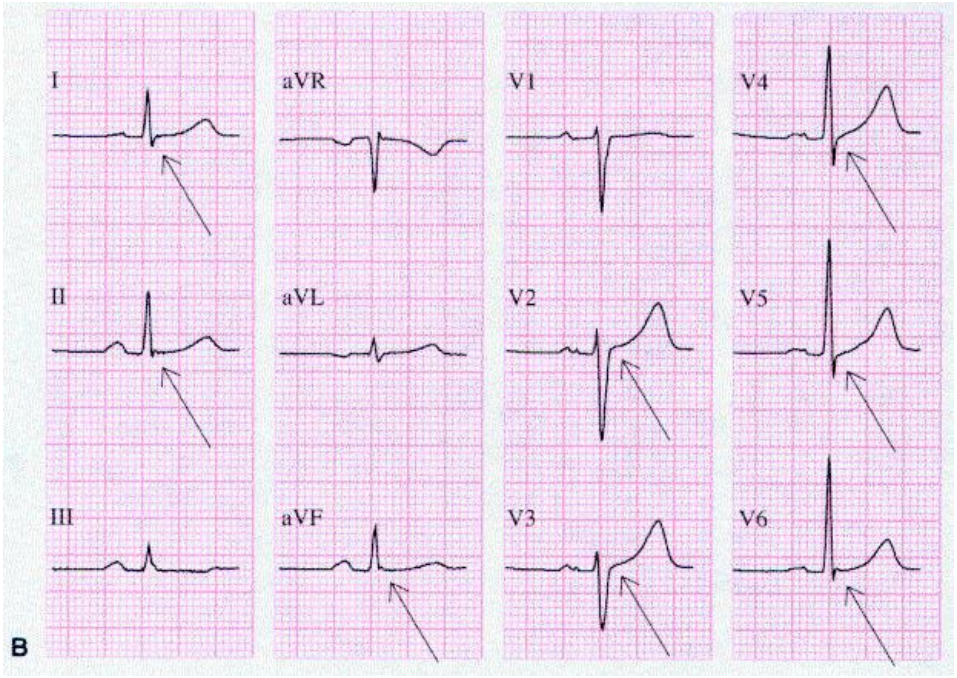
Stages of ECG Evolution in Acute Pericarditis

Stage	PR segments	ST segments	T waves
1	Depressed	Elevated (Diffuse)	Upright
2	Isoelectric	Isoelectric	Flat,upright
3	Isoelectric	Isoelectric	Inverted
4	Isoelectric	Isoelectric	Upright

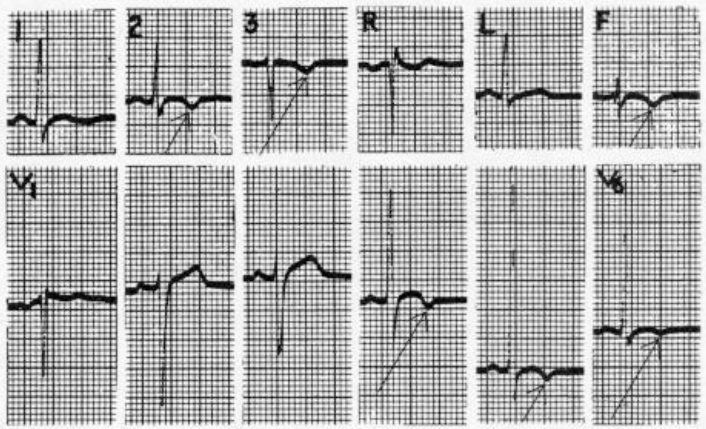
Acute pericarditis – Stage I



Pericarditis-Stage II



Pericarditis-Stage III



T wave inversions

Differential Diagnoses

Clinical

1) Myocardial Infarction
2) Aortic dissection
3) Pulmonary embolism
4) Myocarditis
5) Pneumothorax
6) Musculoskeletal

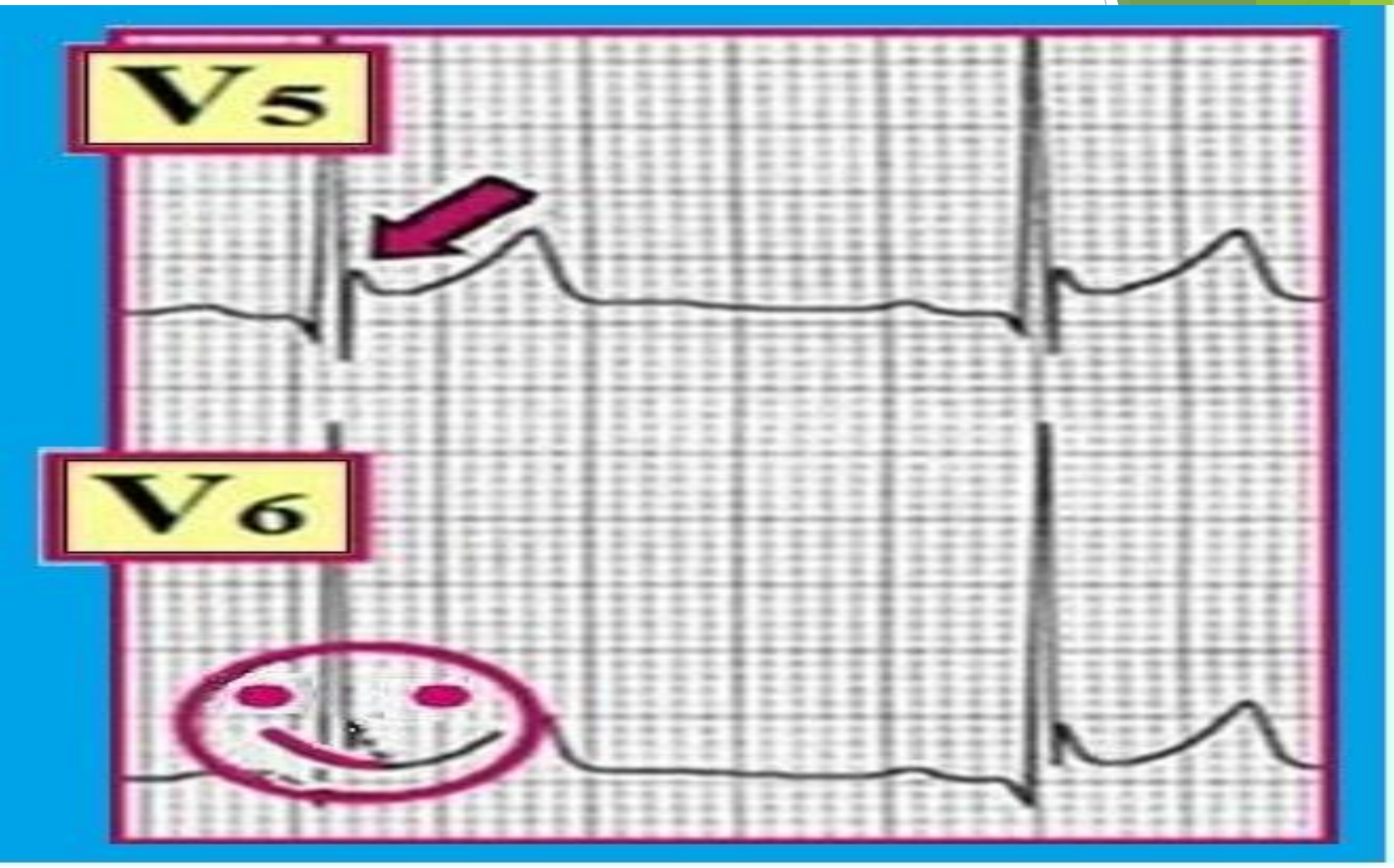
EKG (ST elev)

1) AMI
2) Early Repolarization
3) Myocarditis
4) Hyperkalemia
5) Ventricular Aneurysm
6) Normal Variant

Pericarditis vs Early Repolarization

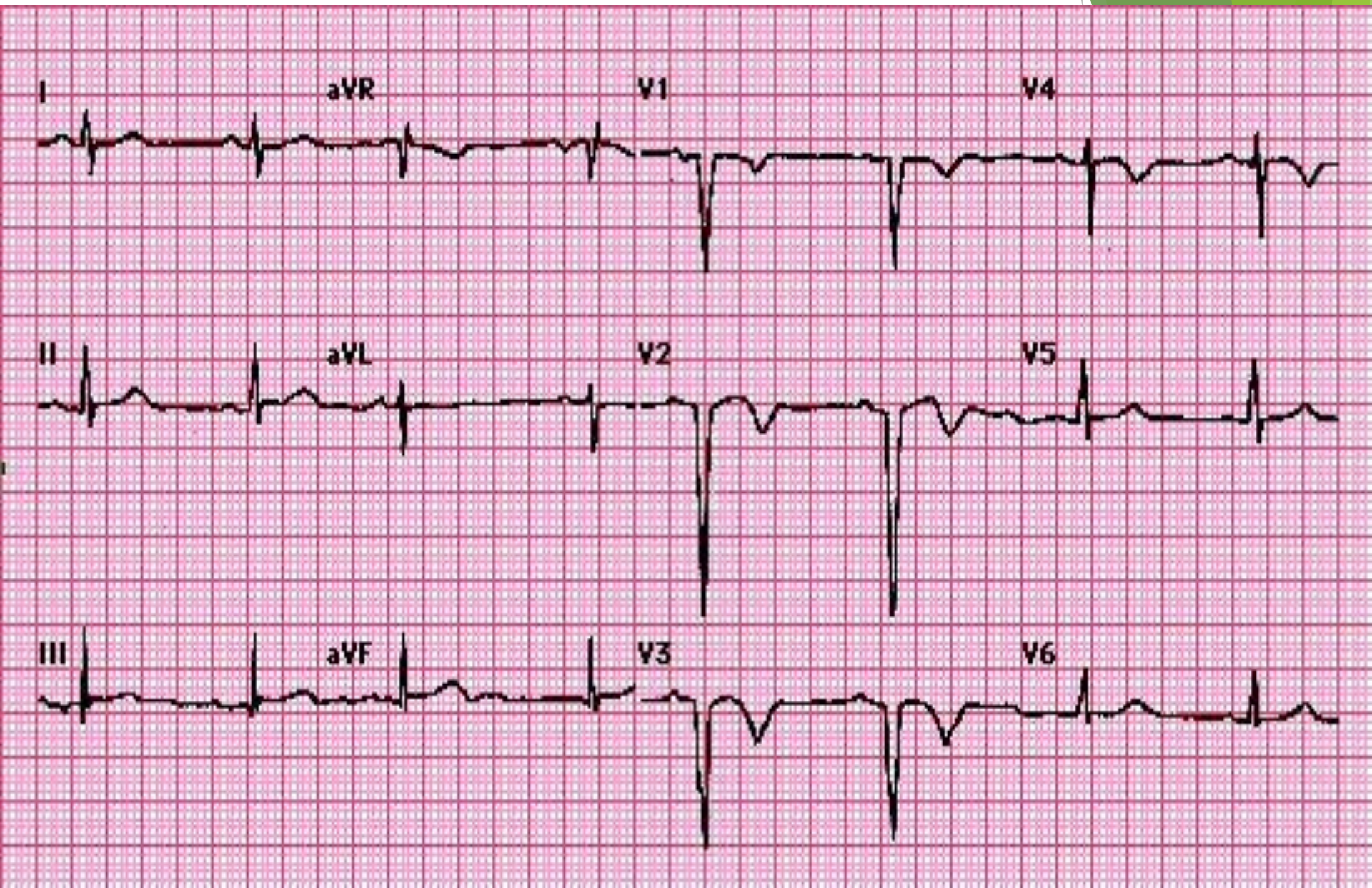
	Acute Pericarditis	Early Repolarization
Sex	Either	Usually Male
Age	Any	Usually < 40
PR segment dev	Common	Uncommon
T waves	inversion, blunt	tall, peaked
J-ST / T ampl V6	> 25%	<25%
Tallest precordial R	Usually V5	Usually V4

Early Repolarization



	Pericarditis	MI
ST segment	• Diffuse,concave elevation in all leads except aVR • reciprocal changes (R,v1) • Height Not > 5mm	• Localized, convex • reciprocal changes in infarct • Height may be > 5 mm
PR depression	• Frequent	• Almost never
Q waves	• Not usual, unless with infarct	• Common with q wave infarct
T waves	• Inverted after J returns to baseline • T inversions and ST ↑ are not seen simultaneously on the same EKG • Hyperacute T waves is rarely seen	• Inverted while ST still elevated • T inversions and ST ↑ can be seen simultaneously on the same EKG • Hyperacute T waves can be seen
Arrhythmias	• Rare	• Frequent
Conduction disturbances	• Rare	• frequent

anterior myocardial infarction



Pericardial complications of myocardial infarction

1) Early infarct-associated pericarditis (often termed peri-infarction pericarditis)
2) Pericardial effusion (with or without tamponade)
3) Postcardiac injury (Dressler's) syndrome

- Each of these complications is related to infarct-size , and has declined in incidence in the revascularization era

PERI-INFARCTION PERICARDITIS (PIP)

- PIP occurs in approximately 5 % of patients treated with fibrinolytic agents versus 20 % in those not receiving fibrinolytic therapy
- ST segments that remain elevated, with persistence of upright T waves, may suggest PIP .
- pericardial rubs were heard on day 1 or 2
- Echocardiography should be performed to evaluate for the presence of a pericardial effusion.
- we do not routinely modify antiplatelet therapy.
- anticoagulation should be immediately discontinued if a pericardial effusion develops or increases
- treatment with routine anti-inflammatory therapy is generally avoided in patients with PIP because of associated risks (hemorrhagic pericardial effusion, scar thinning and infarct expansion).
- The use of corticosteroids after myocardial infarction has been associated with a greater incidence of ventricular aneurysm formation

Which of the following ECG abnormalities is NOT typically associated with acute pericarditis?

1) PR depression
2) Diffuse, concave ST elevation
3) PR elevation in lead aVR
4) Evolution of the Q waves
5) T wave inversion after segment normalization

Which of the following ECG abnormalities is NOT typically associated with acute pericarditis?

1) PR depression
2) Diffuse, concave ST elevation
3) PR elevation in lead aVR
4) Evolution of the Q waves
5) T wave inversion after segment normalization

4- Echocardiogram

- Normal unless there is an effusion
- Presence of effusion supports the diagnosis, but absence does not exclude it.
- Acute pericarditis is accompanied by pericardial effusion in 60% of cases and tamponade in as many as 15% of cases
- The ACC/AHA/ASE all recommend to obtain an echo in any suspected pericardial disease

Laboratory testing

1) CBC – very high WBC (purulent pericarditis)
2) Cardiac enzymes (Troponin , CK-MB)
3) ↑ESR
4) ↑CRP
5) Tuberculin skin test
6) HIV in selected cases
7) ANA
8) Rheumatoid factor
9) Blood cultures if febrile
10) Viral cultures and antibody testing not indicated

❖ **Symptoms and sings :**

- disapperance of cheast pain.
- Pericardial rub might disappear or it may remain
- heart sound become distant and apical impulse not visible.

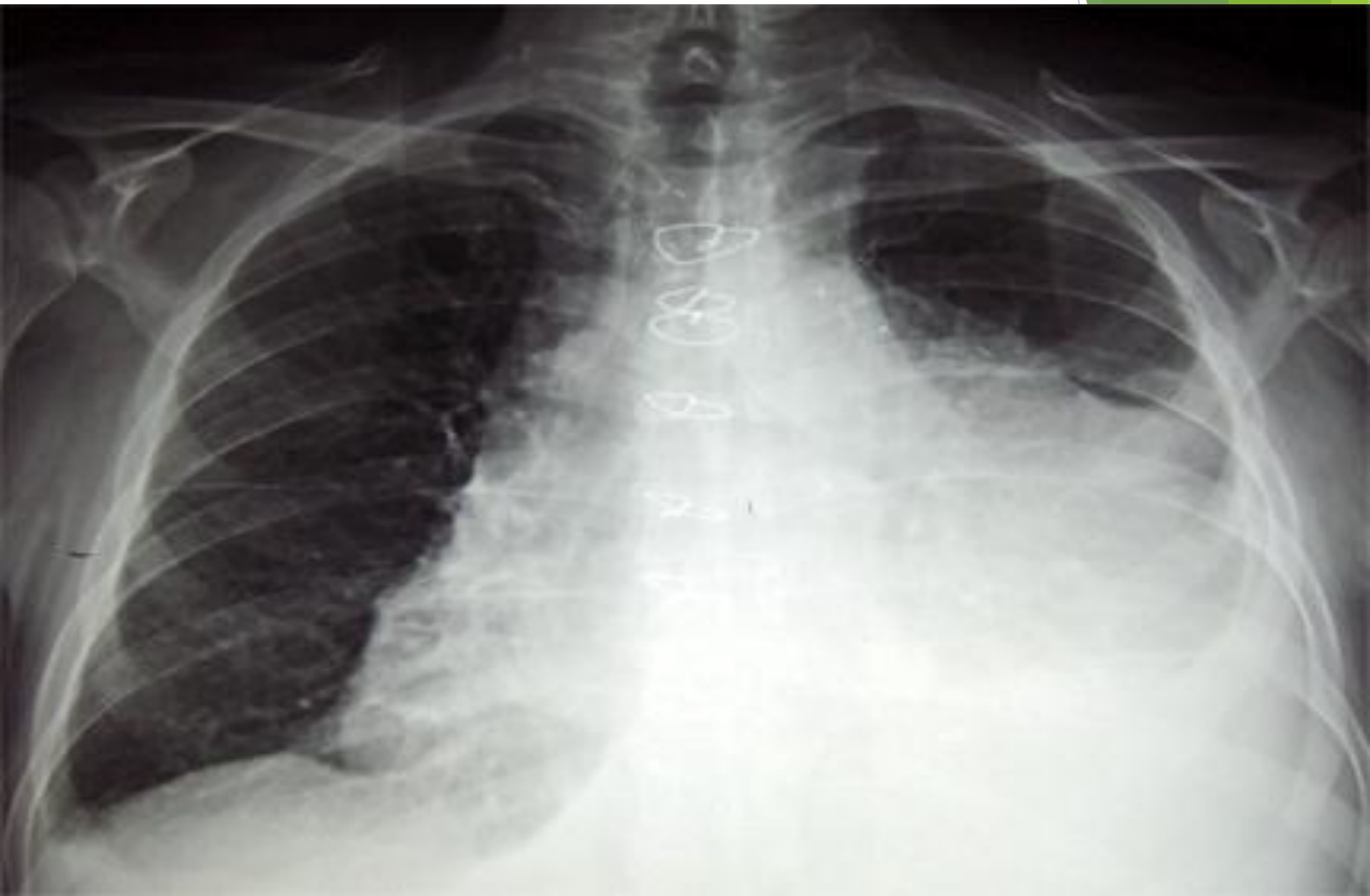
❖ **ECG:**

1) Change in T wave not specific for P. effusion.
2) T wave flat or T wave inverted.
3) Low voltage (The most common) .
4) electrical alternans
5) sinus tachycardia (The combination with low voltage and electrical alternans refers to tamponade) .

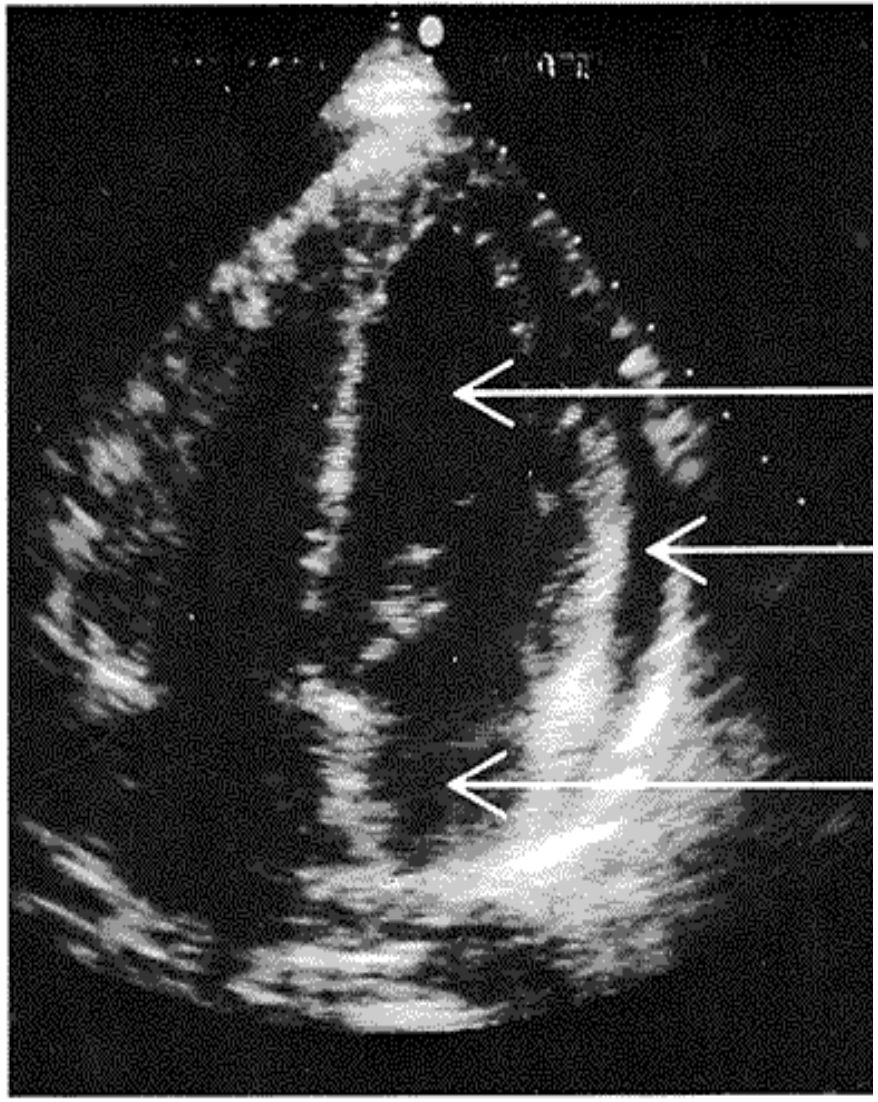
❖ **ECHO:**

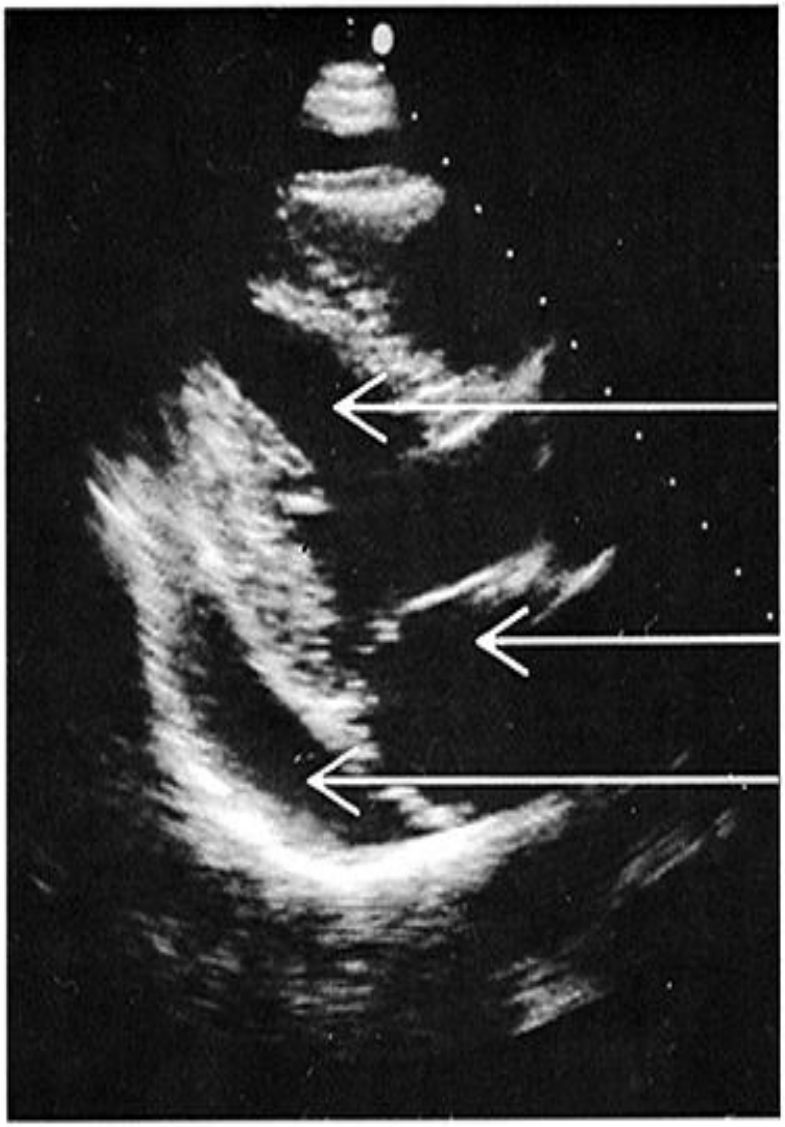
- It is 99% diagnostic to pericardial effusion.
- ECHO: Showed free area between pericardium and posterior wall of L. ventricle.

Cx-Ray in Pericardial Effusion



pericardial effusion





Complication of pericardiatis

1- Cardiac tamponade :

- it is an acute emergency
- It develops When intra pericardial pressure equal to the diastolic pressure in the heart.
- Almost patients with tamponade have one or more of the following physical findings:

1) Hypotension
2) Sinus tachycardia
3) Pulsus paradoxus (it is change in sytolic blood pressure more than 10 mm of Hg during inspiration)
4) Kussmaul's sign (filling of superior vena cava during inspiration)

2- constrictive pericardits :

- whole pericardium is thickened and fibrosis so make thick fibrous layer around heart.
- can be (transient) 10% may have transient within 1st month, resolves by 3 months
- General weakness , wasting , Anaroxia , Dyspnea .
- the history of previous attack of pericarditis is important

3- Recurrent Pericarditis (15-30%):

- > 6 weeks from initiating event
- Most likely an autoimmune etiology
- It is reduced in people who initially take colchicine

Diagnostic criteria for acute and recurrent pericarditis

Acute pericarditis (at least 2 criteria of 4):
Typical pleuritic chest pain
Pericardial friction rub
Suggestive electrocardiogram changes (typically widespread ST segment elevation, PR depression)
New or worsening pericardial effusion
Recurrent pericarditis
Typical pleuritic chest pain plus at least one objective evidence of disease activity:
Fever
Pericardial rub
ECG changes
New or worsening pericardial effusion
Elevation of markers of inflammation (elevation in white blood cell count, erythrocyte sedimentation rate, or C-reactive protein)

Indications for invasive workup in acute pericarditis

Pericardiocentesis:
1. Cardiac tamponade
2. Moderate to large effusions refractory to medical therapy and with severe symptoms
3. Suspected bacterial or neoplastic pericarditis
Pericardial biopsy and pericardioscopy (targeted biopsy in specialized center):
1. Relapsing cardiac tamponade
2. Suspected bacterial or neoplastic pericarditis
3. Worsening pericarditis (despite medical therapy) without a specific diagnosis

Need for hospitalization

- Many physicians tend to admit them, but this may not be necessary.
- Features of high risk include:
  1) Subacute symptoms (eg, developing over several days or weeks)
  2) High fever (>38ºC [100.4ºF]) and leukocytosis
  3) Evidence suggesting cardiac tamponade
  4) A large pericardial effusion
  5) Immunosuppressed state
  6) A history of oral anticoagulant therapy
  7) Acute trauma
  8) Failure to respond within seven days to NSAID therapy, a generous allocation of time
  9) Elevated cardiac troponin, suggestive of myopericarditis

Treatment

- **Goals of acute therapy:**
  1) Relieve Pain
  2) Treat the inflammation
  3) Prevent Cardiac tamponade and constrictive P .
- Most viral infections are self-limited
- Treat the underlying disease process
- Drain purulent effusions
- Symptomatic therapy
- None of the treatments unfortunately, have not been proven to prevent the complications.

NSAIDs

- May require weeks to months of treatment with high doses of NSAIDs , and relieved CP in 85-90% of patients
- If adequate clinical response,continue NSAIDs for 1 wk after complete resolution of Sx and then taper in 2–3 days.

1- Aspirin

- 650 to 1000 mg three times daily
- Preferred in patients with MI

2- Ibuprofen

- 600 to 800 mg three times daily
- above average response rate and has a very good side effect profile

3- Indomethacin

- 50 mg three times daily
- Try to avoid, it can ↓ coronary blood flow.

Colchicine (Colcrys ®)

- NSAIDs have not been shown to prevent tamponade or long term complications such as constrictive pericarditis or recurrent pericarditis.
- A prospective, randomized, open-label design was used.
- 120 patients with a first episode of acute pericarditis were randomly assigned to
  - conventional treatment with aspirin (group I) or
  - conventional treatment plus colchicine 1.0 to 2.0 mg for the first day and then 0.5 to 1.0 mg/d for 3 months (group II).
  - **Colchicine significantly reduced the recurrence rate** (10.7% vs 32.3%; *P*=0.004;) and presence of symptoms at 72 hours (11.7% vs 36.7%; *P*=0.003).
  - Based upon this, addition of it to the Rx regimen for an initial episode of acute pericarditis is an option for physicians

Treatment of PIP

- ACC/AHA guidelines recommend aspirin as the preferred agent for the treatment of PIP, noting that doses as high as 650 to 1000 mg three times daily may be needed. (I)
- colchicine (0.5 mg every 12 hours) (IIa)
- corticosteroids or non-steroidal anti-inflammatory agents (IIb)
- Ibuprofen (III harm)

Steroids

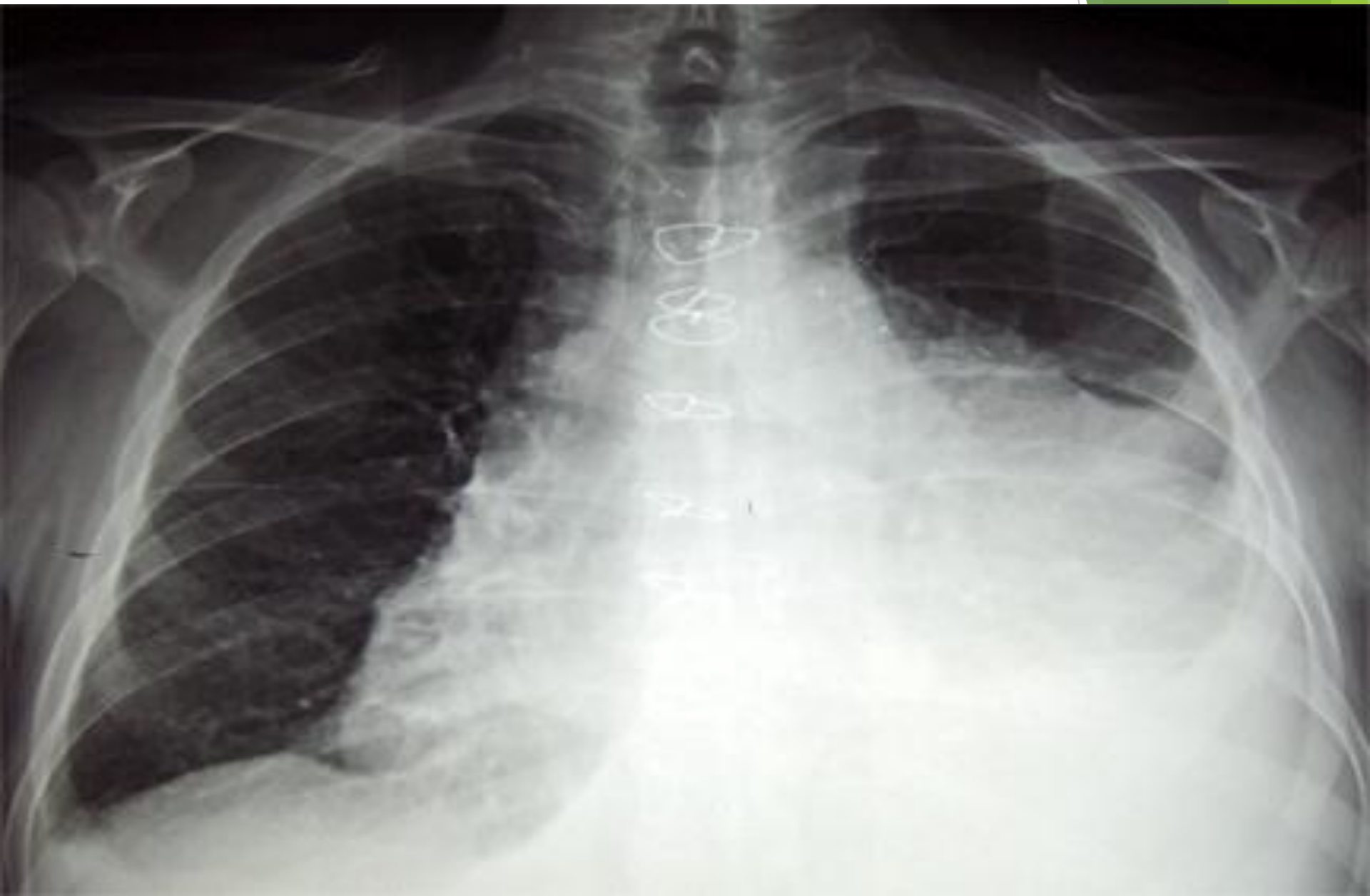
- In patients refractory to NSAIDs and colchicine or cannot be tolerated
- Steroid therapy with initial episode is more likely associated with recurrent episodes.
- Evidence available argues against the routine administration of corticosteroids during a first episode of acute pericarditis
- Specific conditions that will benefit:
    1) Acute pericarditis due to connective tissue diseases
    2) Auto-immune pericarditis
    3) Uremic pericarditis

Drug therapy in acute pericarditis for adult patients

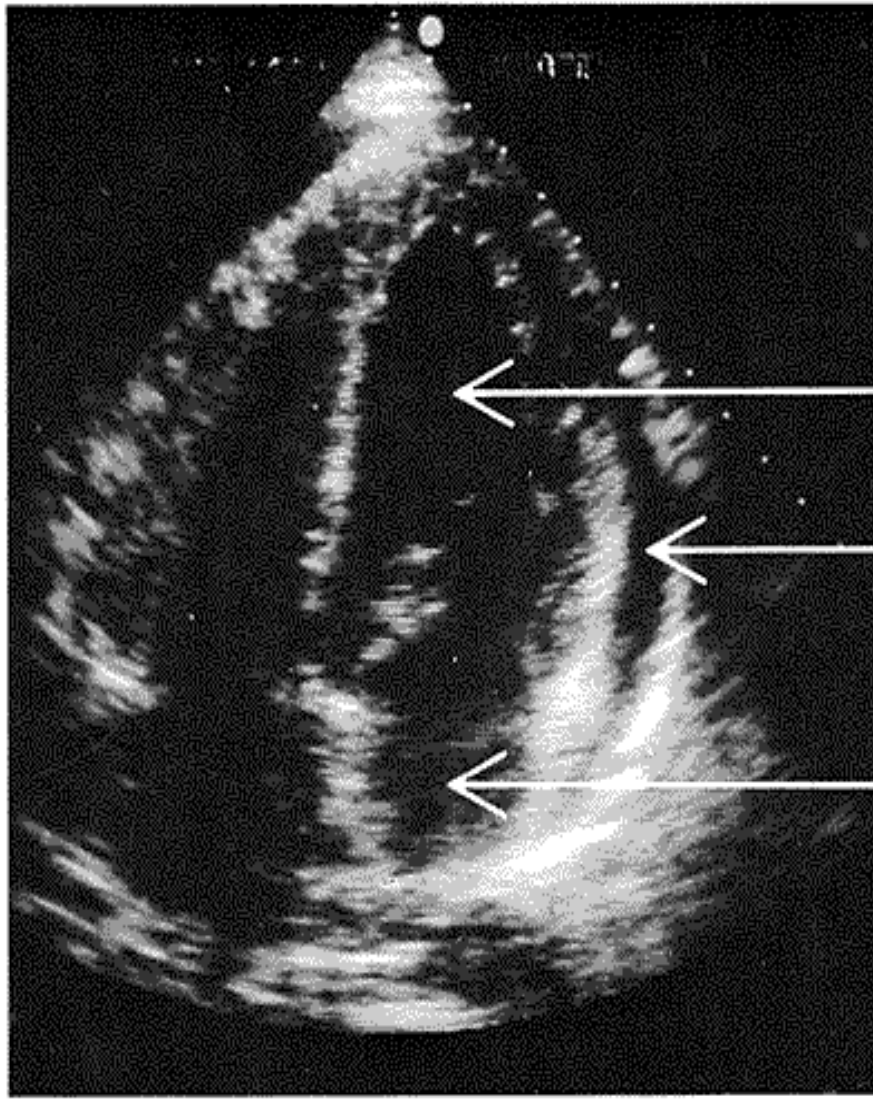
Drug	Dose*	Duration of therapy
For initial combination treatment of most patients:		
Ibuprofen •	600 to 800 mg three times daily	1 to 2 weeks
OR		
Indomethacin •	50 mg three times daily	1 to 2 weeks
PLUS		
Colchicine Δ§	0.5 to 0.6 mg two times daily	3 months
For initial combination therapy of patients following myocardial infarction:		
Aspirin •	650 to 1000 mg three times daily	1 to 2 weeks
PLUS		
Colchicine Δ§	0.5 to 0.6 mg two times daily	3 months
For refractory cases or patients with a contraindication to NSAID therapy:		
Prednisone	0.25 to 0.5 mg/kg/day	2 weeks
PLUS		
Colchicine Δ§	0.5 to 0.6 mg two times daily	3 months ¥

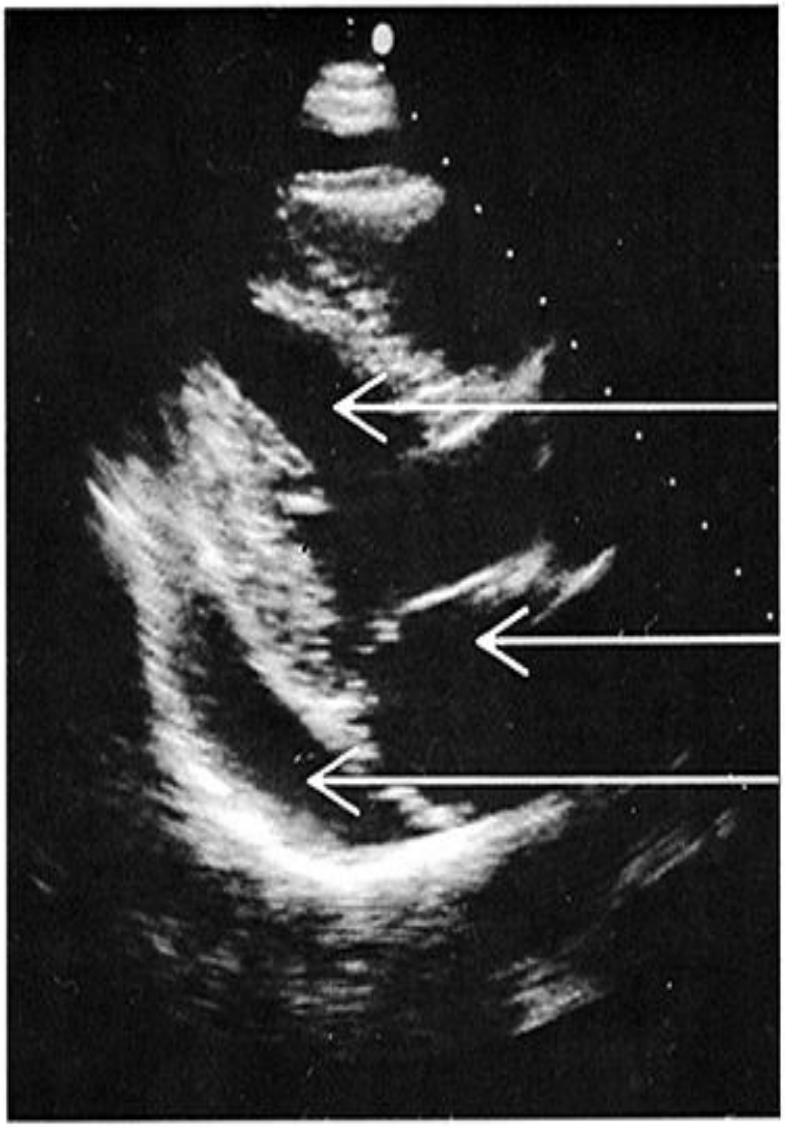
2- Precardial effusion without temponade

Cx-Ray in Pericardial Effusion



pericardial effusion





Pericardial Effusion

Normal 15-50 ml of fluid

ETIOLOGY

1. Inflammation from infection, immunologic process.
2. Trauma causing bleeding in pericardial space.
3. Noninfectious conditions such as:

a. increase in pulmonary hydrostatic pressure e.g. heart failure. congestive

b. increase in capillary permeability e.g. hypothyroidism

c. decrease in plasma oncotic pressure e.g. cirrhosis.

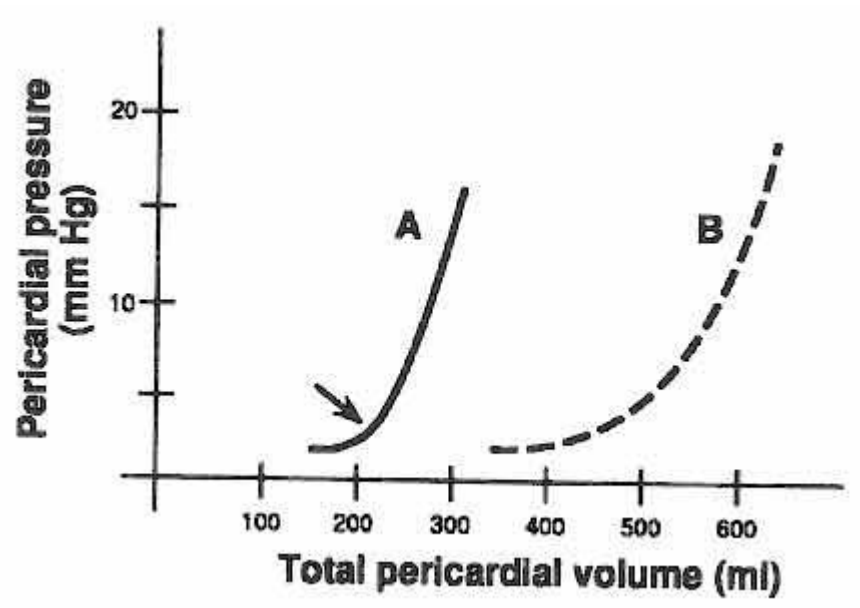
4. Decreased drainage of pericardial fluid due to obstruction of thoracic duct as a result of malignancy or damage during surgery.

- Effusion may be serous, serofibrinous, suppurative, chylous, or hemorrhagic depending on the etiology.

Pathophysiology

Pericardium relatively stiff
Symptoms of cardiac compression dependant on:
1. Volume of fluid
2. Rate of fluid accumulation
3. Compliance characteristics of the pericardium

A. Sudden increase of small amount of fluid (e.g. trauma)
B. Slow accumulation of large amount of fluid (e.g. CHF)



Clinical features

Small effusions do not produce hemodynamic abnormalities.

Large effusions, in addition to causing hemodynamic compromise, may lead to compression of adjoining structures and produce symptoms of:
dysphagia (compression of esophagus)
hoarseness (recurrent laryngeal nerve compression)
hiccups (diaphragmatic stimulation)
dyspnea (pleural inflammation/effusion)

Physical Findings

Physical Findings:

Muffled heart sounds

Paradoxically *reduced* intensity of rub

Ewart's sign:

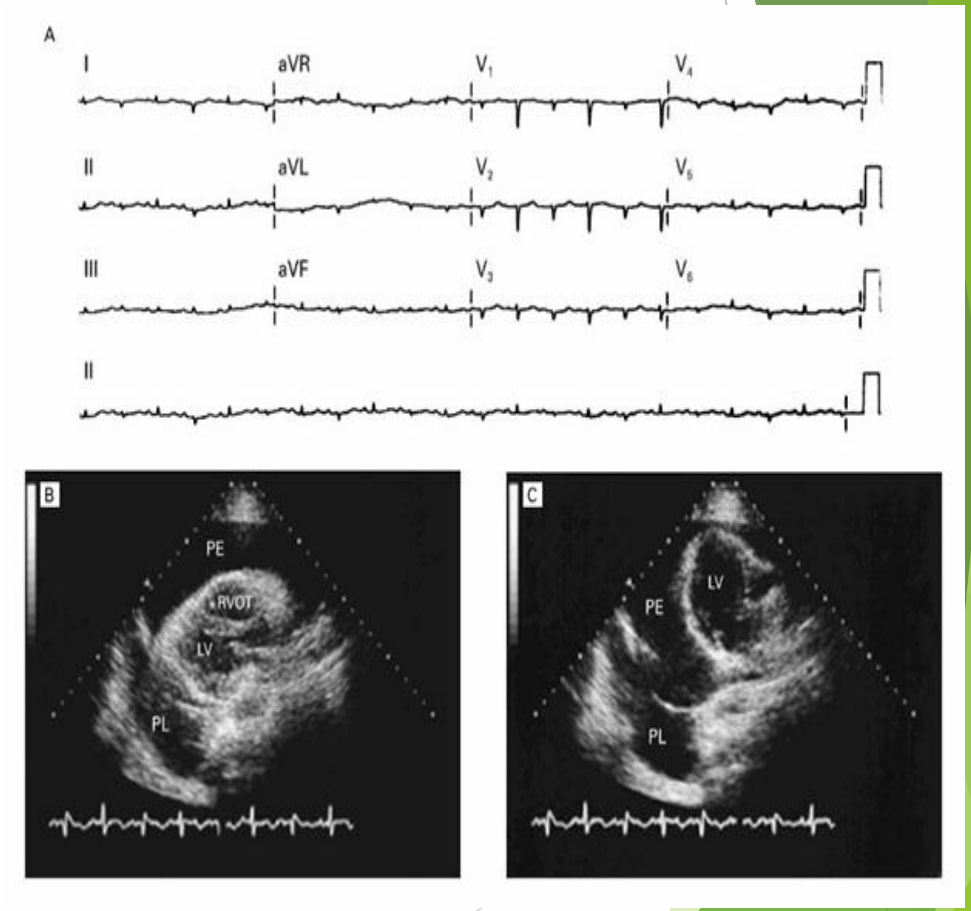
Compression of lung leading to an area of consolidation in the left infrascapular region (atalectasis, detected as dullness to percussion and bronchial breathing)

Diagnostic studies

CXR: "water bottle" shaped heart

EKG:
low voltage➢
"electrical alternans"➢

Echocardiogram



Pericardial Effusion

- Accumulation of fluid in the pericardial sac (between the parietal and visceral layers of the serous pericardium) beyond the normal physiologic amount
- Types: serous, fibrinous, purulent, or hemorrhagic

ETIOLOGY

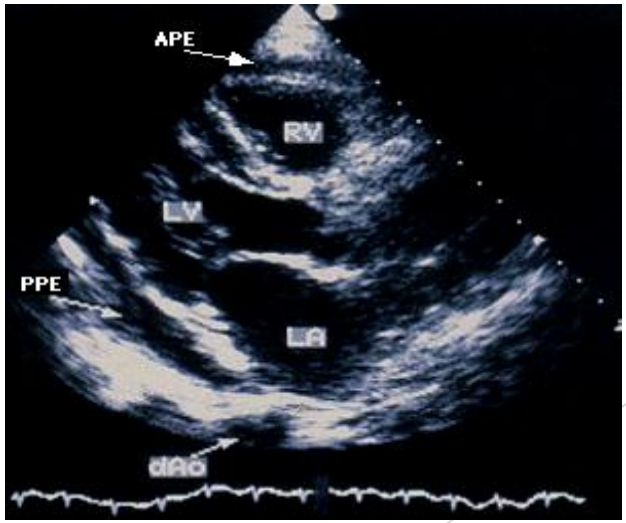
- Heart/renal/liver failure
- Infection/inflammation
- Neoplasm
- Myocardial infarction
- Trauma

ROLE OF MRI

- Diagnosis of effusion
- Quantification of effusion
- Characterize nature of fluid (transudate vs exudate)
- Hemodynamic consequences (tamponade/ constriction)
- Associated inflammation
- Guidance for pericardiocentesis

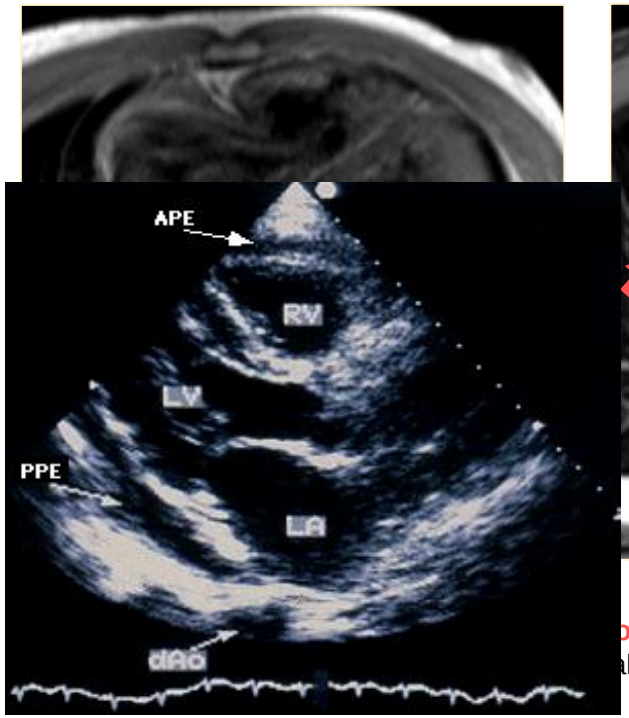
Pericardial Effusion

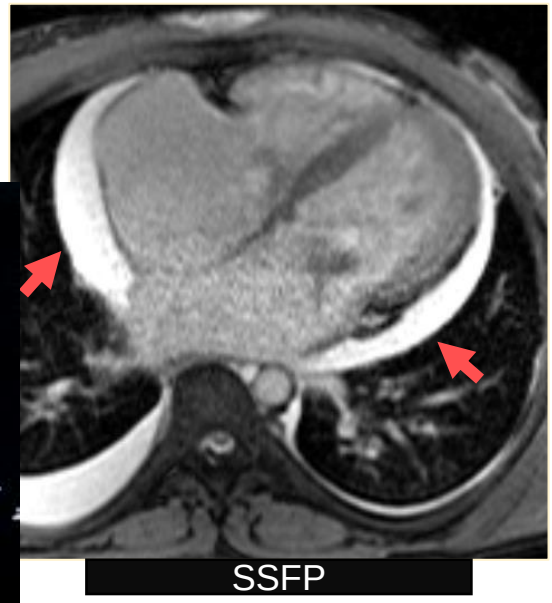
- **MRI** is more sensitive than echocardiography for small collections, especially for loculated effusion.
- No clear correlation between pericardial space thickness and volume of pericardial fluid.
- **Early phase:** accumulates posterolateral to left ventricle due to gravity; then in the superior recess
- **Moderate (100-500 ml):** > 5-mm pericardial space anterior to the right ventricle
- However, dimensional association with overall pericardial effusion size is challenging, especially with loculated effusions or with regional variations.
- The rate of fluid accumulation is more important than the size of the effusion.
- The pericardium could stretch and contain much larger volumes without any clinical symptoms *when the fluid is slowly accumulated.*



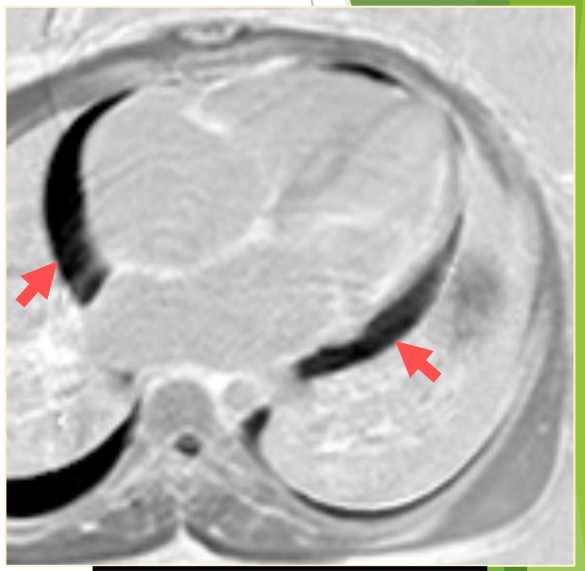
Pericardial Effusion: Simple

- Low signal intensity on T1-weighted images and high signal intensity on T2-weighted fast spin-echo (FSE) and SSFP images
- Dark signal on black blood and phase-sensitive inversion-recovery (PSIR) delayed enhancement images





SSFP



PSIR

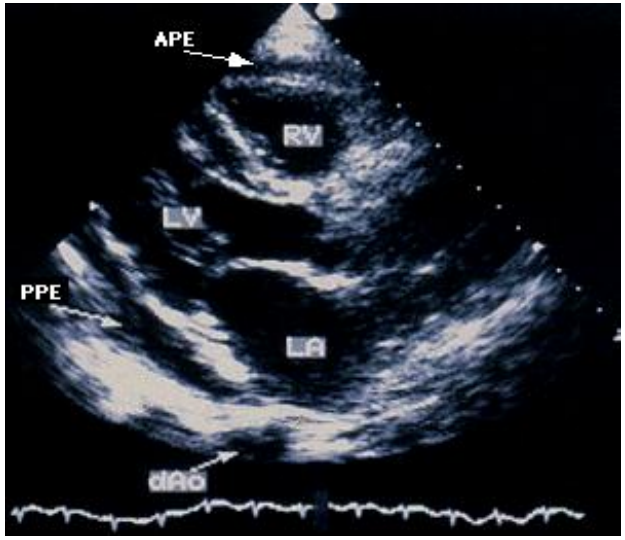
ount of circumferential clear pericardial fluid (arrows):
l intensity; ***SSFP:*** bright signal intensity; ***PSIR:*** dark signal intensity

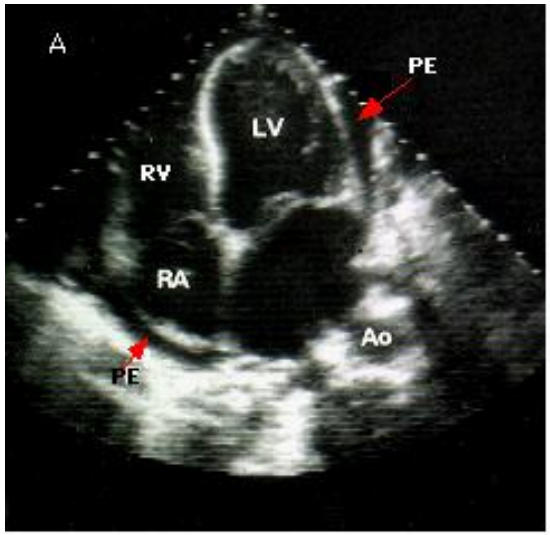
Cardiac Tamponade

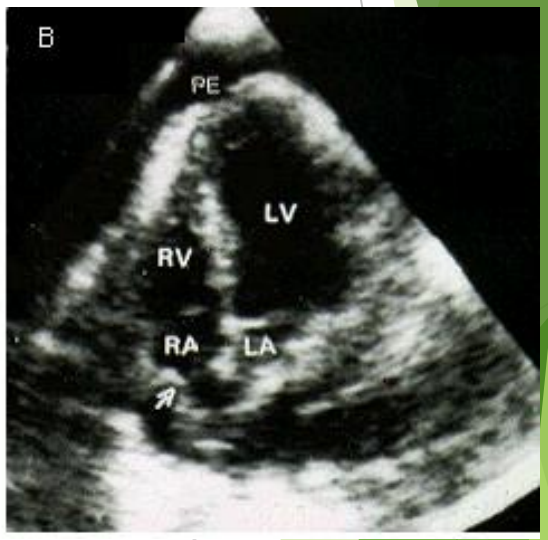
Fluid under high pressure compresses the cardiac chambers:

acute: trauma, LV rupture – may not be very large

gradual: large effusion, due to any etiology of acute pericarditis

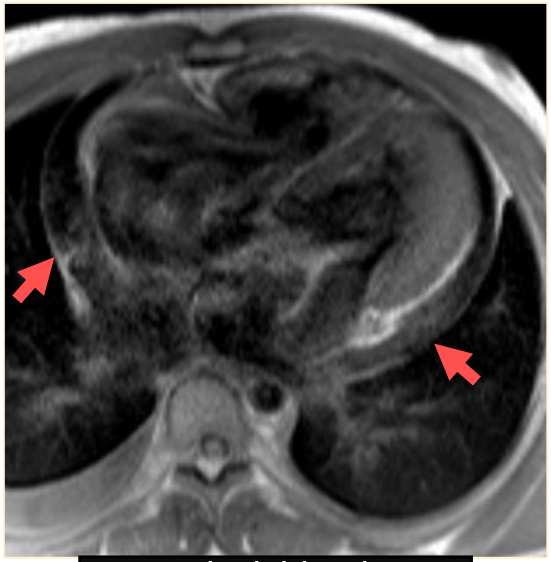




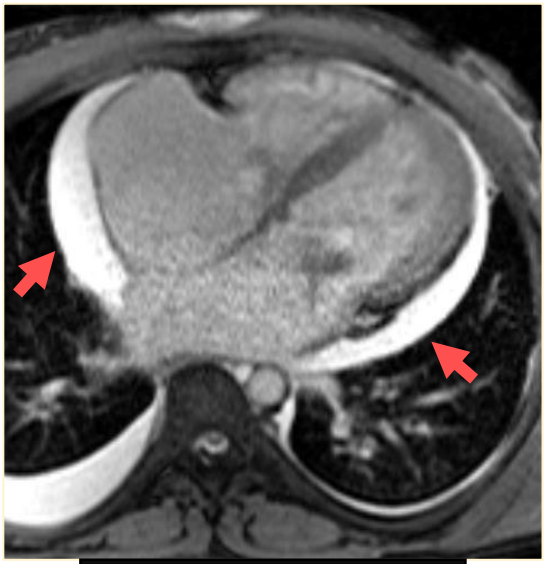


Pericardial Effusion: Simple

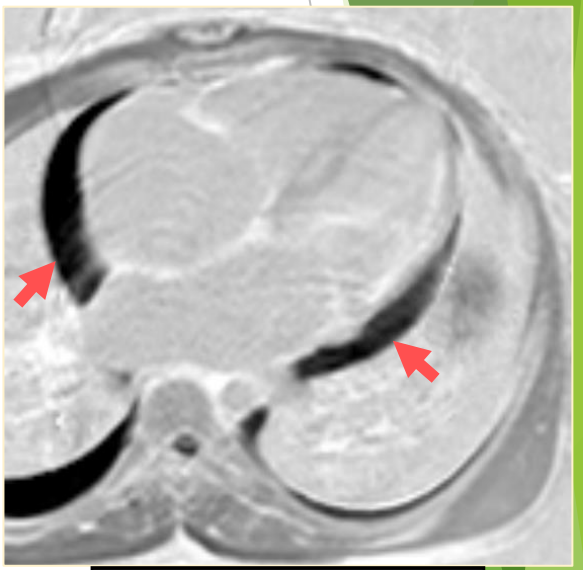
- Low signal intensity on T1-weighted images and high signal intensity on T2-weighted fast spin-echo (FSE) and SSFP images
- Dark signal on black blood and phase-sensitive inversion-recovery (PSIR) delayed enhancement images



Black blood



SSFP



PSIR

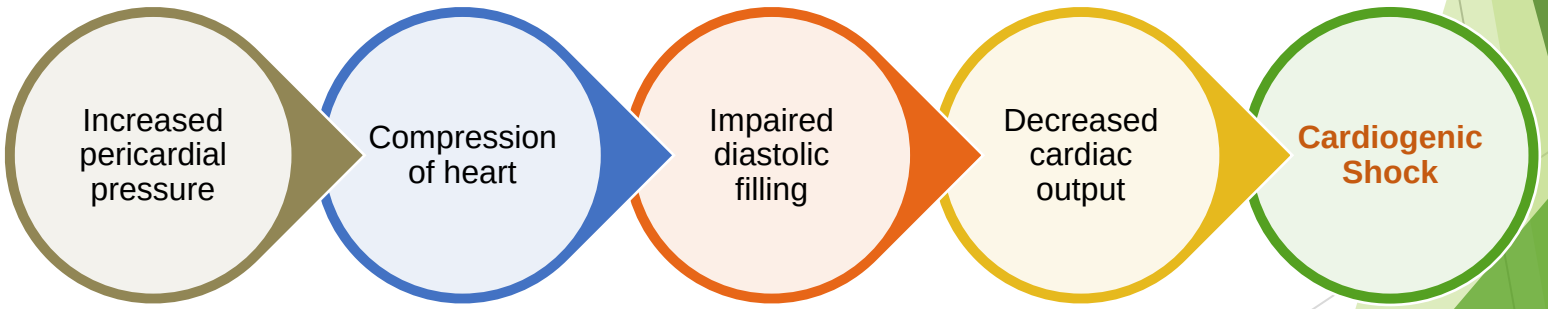
Moderate amount of circumferential clear pericardial fluid (arrows):
Black blood: dark signal intensity; ***SSFP:*** bright signal intensity; ***PSIR:*** dark signal intensity

Cardiac Tamponade

- Pericardial accumulation of fluid, blood, pus, or gas that results in impaired cardiac output
- It may occur rapidly (acute) or gradually (chronic)
- Pericardial effusion size does not necessarily correspond to tamponade physiology
- The rate of accumulation is more important than the total amount of the fluid

Etiology

- Ruptured myocardial infarction
- Ruptured aneurysm
- Dissection
- Thrombolysis
- Blunt chest injury
- Coronary artery bypass grafting (CABG) surgery
- Endocarditis
- Tumors



Pathophysiology

Tamponade-- Clinical Features

Symptoms:
Acute: (trauma, LV rupture)
profound hypotension
confusion/agitation
Slow/Progressive large effusion (weeks)
Fatigue (↓CO)
Dyspnea
JVD

Signs:
Tachycardia
Hypotension
rales/edema/ascites
muffled heart sounds
pulsus paradoxus

Beck's Triad in Cardiac tamponade

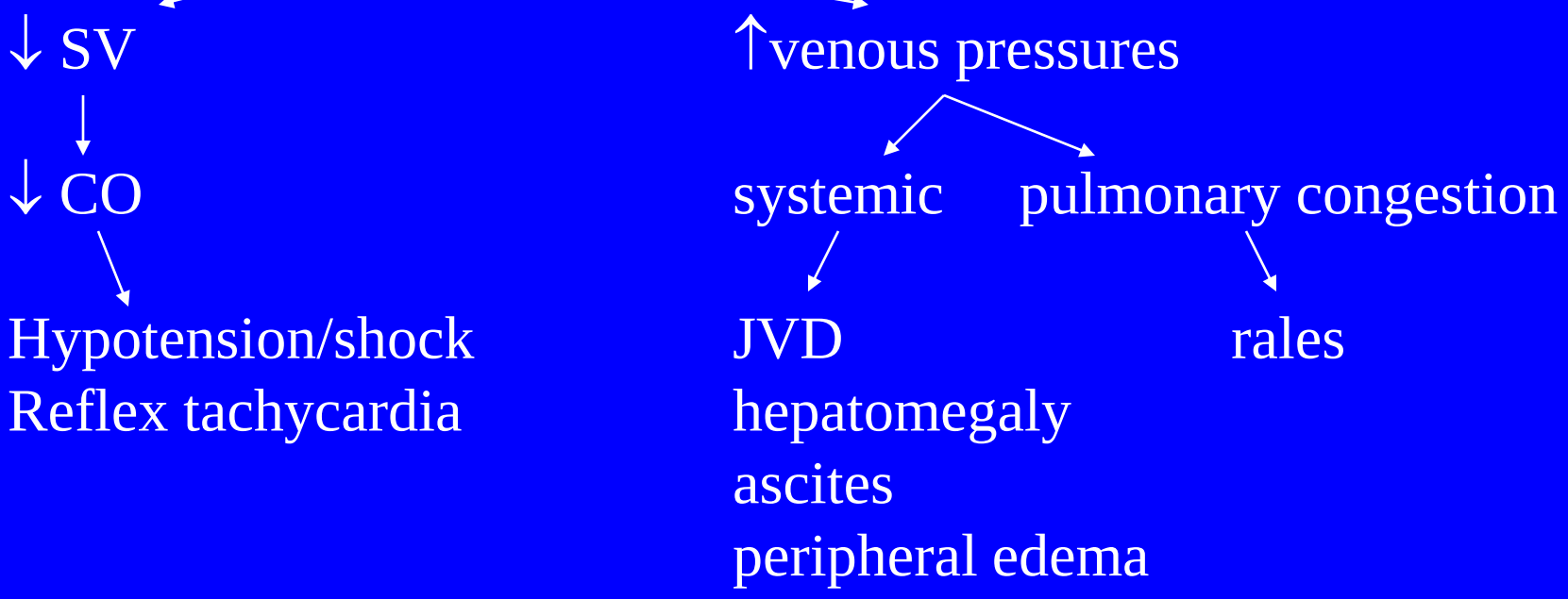
1) Hypotension

2) Jugular venous distension (Kussmal's sign)

3) Distant heart sounds

CardiacTamponade -- Pathophysiology

Accumulation of fluid under high pressure:
compresses cardiac chambers & impairs diastolic filling of **both** ventricles



Pulsus Paradoxus

Intrapericardial pressure (IPP) tracks intrathoracic pressure.

Inspiration:

negative intrathoracic pressure is transmitted to the pericardial space

↓ IPP

↑ blood return to the right ventricle

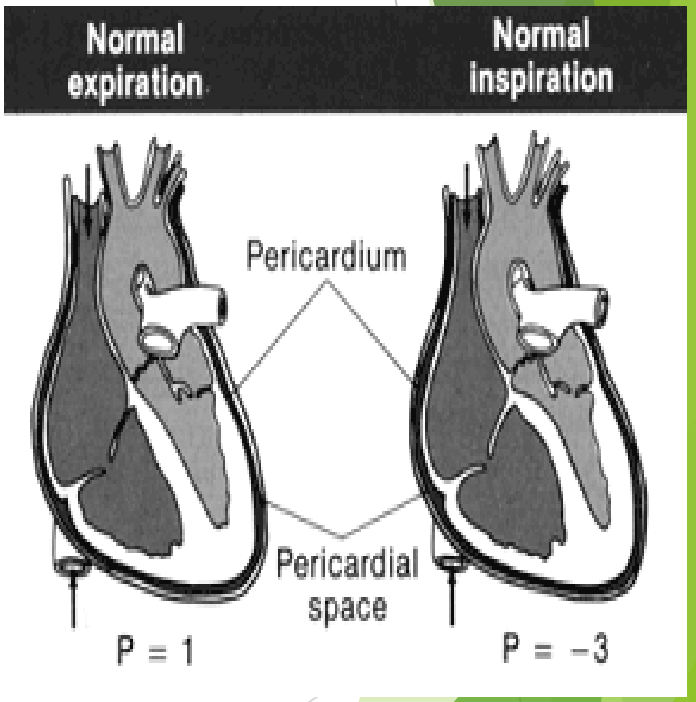
↓ jugular venous and right atrial pressures

↑ right ventricular volume → interventricular septum shifts towards the left ventricle

↓ left ventricular volume

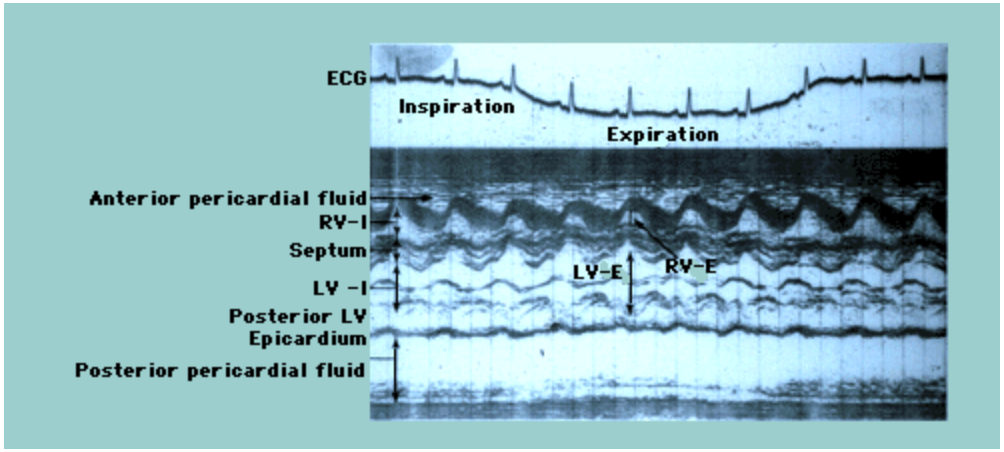
↓ LV stroke volume

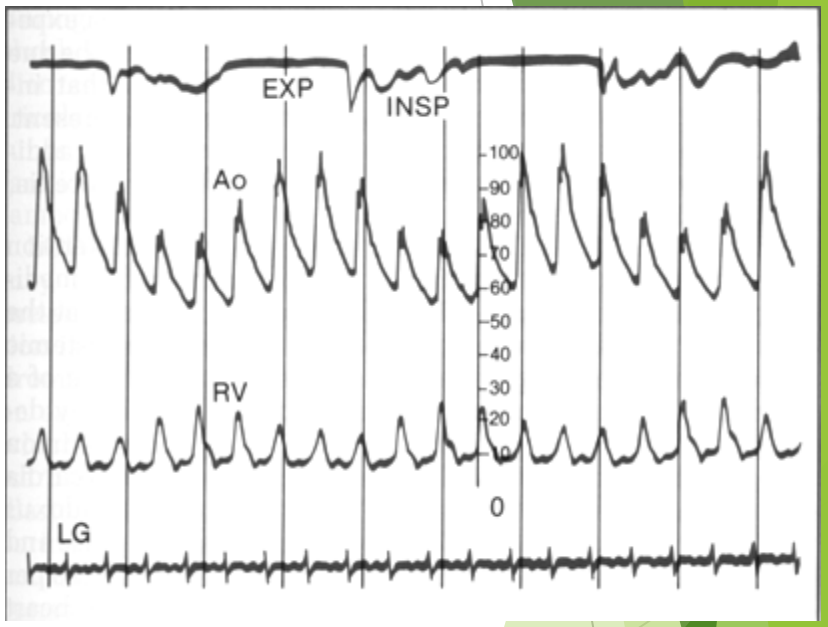
⇒ ↓ blood pressure (<10mmHg is normal) during inspiration



Pulsus Paradoxus

Exaggeration of normal physiology



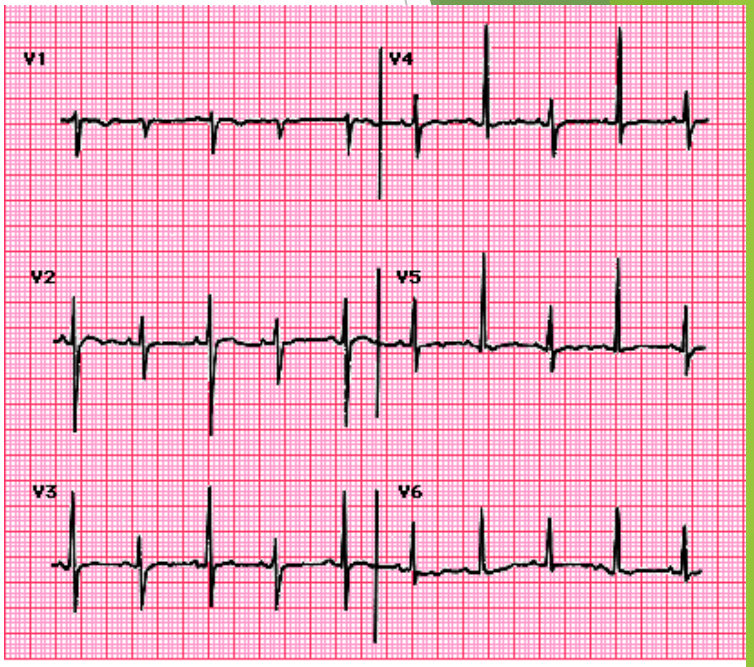


> 10 mm Hg drop in BP with inspiration

Tamponade -- Diagnosis

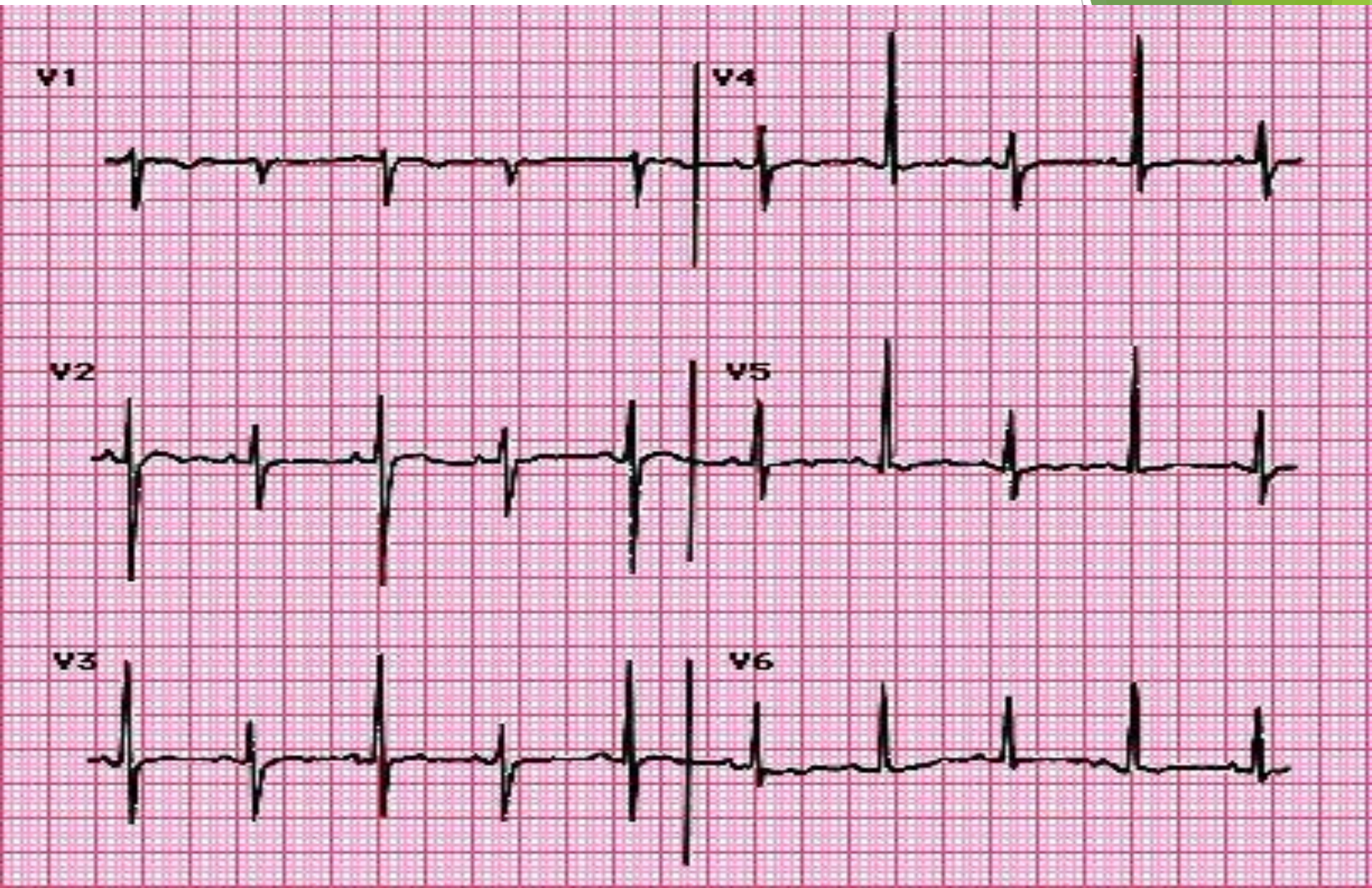
EKG: low voltage, sinus tachycardia,
electrical alternans

Echocardiography
pericardial effusion
(r/o other etiologies in dif dx)
RA and RV diastolic collapse



Electrical alternans Sinus tachycardia with electrical alternans which is characterized by beat-to-beat alternation in the QRS appearance (best seen in leads V2 to V4). These findings are strongly suggestive of pericardial effusion, usually with tamponade. The alternating ECG pattern is related to back-and-forth swinging motion of the heart in the pericardial fluid. Courtesy of Ary Goldberger, MD.

Electrical alternans in Tamponade



Right Heart Catheterization

Catheterization Findings:

Elevated RA and RV diastolic pressures
Equalized diastolic pressures
Blunted “y” descent in RA tracing
y descent: early diastolic filling (atrial emptying)
↓ BP and Pulsus paradoxus
Pericardial pressure = RA pressure

Constrictive Pericarditis

Late complication of pericardial disease
Fibrous scar formation
Fusion of pericardial layers
Calcification further stiffens pericardium
Etiologies:
any cause of pericarditis
idiopathic
post-surgery
tuberculosis
radiation
neoplasm

Pathophysiology

Rigid, scarred pericardium encircles heart:
Systolic contraction normal
Inhibits diastolic filling of **both** ventricles

Physical exam

↑HR, ↓BP

ascites, edema, hepatomegaly

early diastolic “knock”
after S2
sudden cessation of ventricular diastolic filling imposed
by rigid pericardial sac

Kussmaul’s sign

Kussmaul's Sign

inspiration: ↓intrathoracic pressure, ↑ venous return to thorax

↓intrathoracic pressure not transmitted though to RV

⇒ no pulsus paradoxus!

no inspiratory augmentation of RV filling (rigid pericardium)

intrathoracic systemic veins become distended

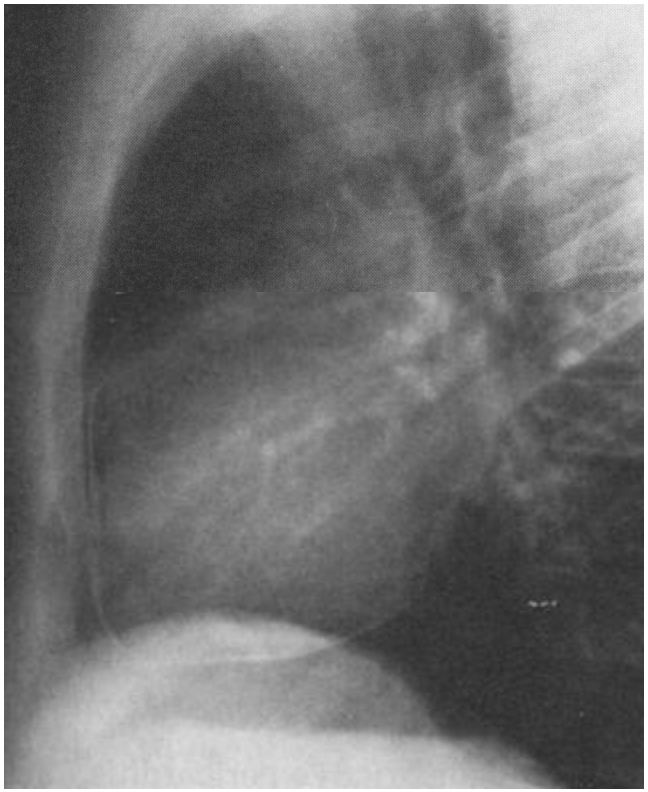
⇒JVP rises with inspiration (normally falls)

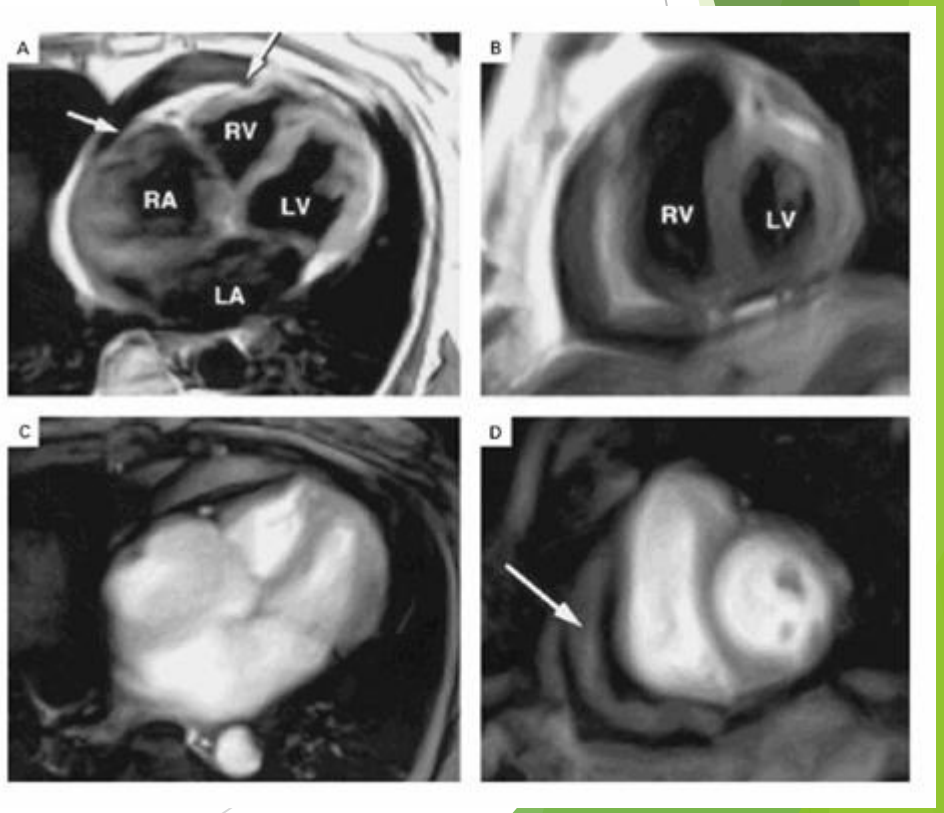
Diagnosis

CXR: calcified cardiac silhouette

EKG: non-specific

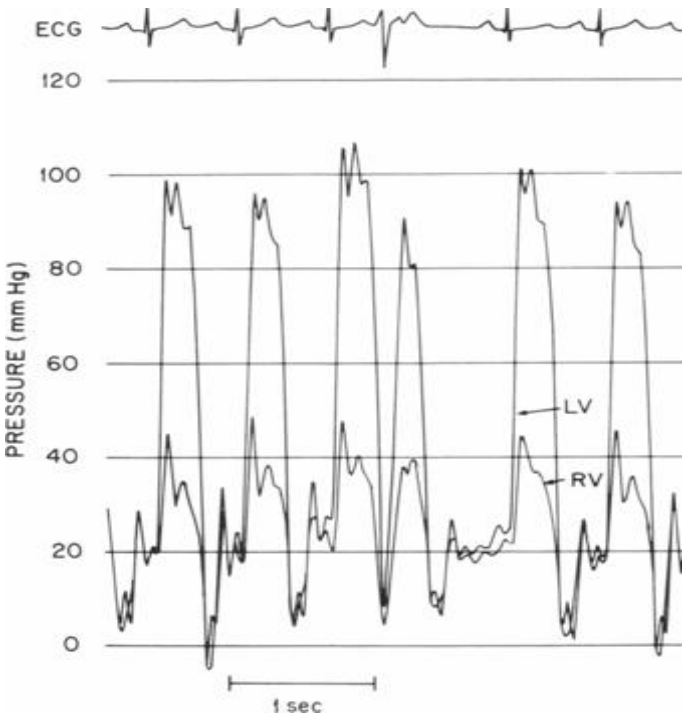
CT or MRI: pericardial thickening

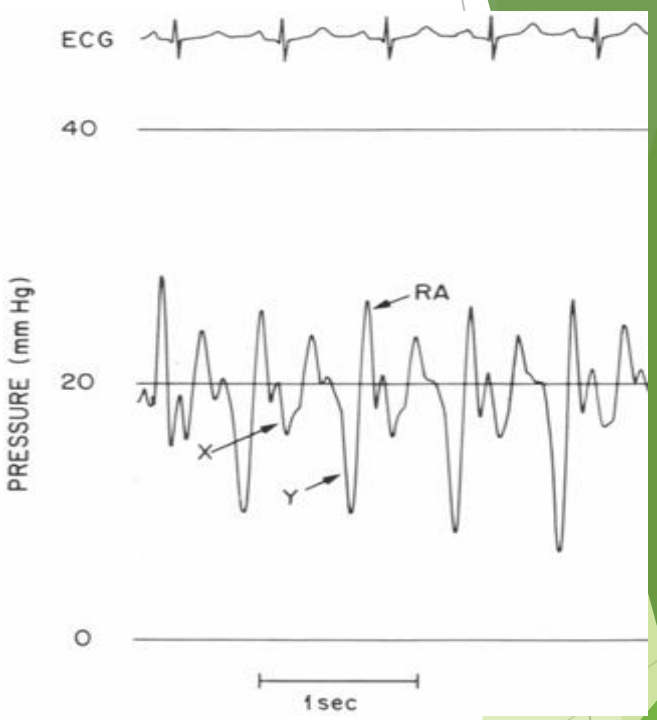




Cardiac Catheterization

Elevated and equalized diastolic pressures (RA=RVEDP=PAD=PCV





"dip and

rapid ventricular

Prominent y descent: plateau": rapid atrial emptying filling

Constriction vs. Restriction

THE DIFFERENTIAL DIAGNOSIS OF RESTRICTIVE CARDIOMYOPATHY AND CONSTRICTIVE PERICARDITIS.*

TYPE OF EVALUATION	RESTRICTIVE CARDIOMYOPATHY	CONSTRICTIVE PERICARDITIS
Physical examination	Kussmaul's sign may be present Apical impulse may be prominent S3 may be present, rarely S4 Regurgitant murmurs common	Kussmaul's sign usually present Apical impulse usually not palpable Pericardial knock may be present Regurgitant murmurs uncommon
Electrocardiography	Low voltage (especially in amyloidosis), pseudoinfarction, left-axis deviation, atrial fibrillation, conduction disturbances common	Low voltage (<50 percent)
Echocardiography	Increased wall thickness (especially thickened interatrial septum in amyloidosis) Thickened cardiac valves (amyloidosis) Granular sparkling texture (amyloid)	Normal wall thickness Pericardial thickening may be seen Prominent early diastolic filling with abrupt displacement of interventricular septum
Doppler studies	Decreased RV and LV velocities with inspiration Inspiratory augmentation of hepatic-vein diastolic flow reversal Mitral and tricuspid regurgitation common	Increased RV systolic velocity and decreased LV systolic velocity with inspiration Expiratory augmentation of hepatic-vein diastolic flow reversal
Cardiac catheterization	LVEDP often >5 mm Hg greater than RVEDP, but may be identical	RVEDP and LVEDP usually equal RV systolic pressure <50 mm Hg RVEDP >one third of RV systolic pressure
Endomyocardial biopsy	May reveal specific cause of restrictive cardiomyopathy	Maybe normal or show nonspecific myocyte hypertrophy or myocardial fibrosis
CT/MRI	Pericardium usually normal	Pericardium may be thickened

*LV denotes left ventricular, RV right ventricular, LVEDP left ventricular end-diastolic pressure, RVEDP right ventricular end-diastolic pressure, CT computed tomography, and MRI magnetic resonance imaging.

Similar presentation and physiology, important to differentiate as constriction is treatable by pericardiectomy

Majority of diseases causing restriction are not treatable

Constriction vs. Tamponade
Summary

TAMPONADE	CONSTRICTION
Low cardiac output state	Low cardiac output state
JVD present	JVD present
NO Kussmaul's sign	Kussmaul's sign
Equalized diastolic pressures	Equalized diastolic pressures
RA: blunted y descent	RA: rapid y descent
Decreased heart sounds	Pericardial "knock"

THANK YOU